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SOME BYGONE OPERATIONS IN SURGERY.

By Sir D'ARCY POWER, K.B.E., London.

IX. A CASE OF STRANGULATED UMBILICAL HERNIA. QUEEN CAROLINE OF ANSPACH.

CAROLINE OF ANSPACH (1683–1737), wife of George II and Queen of England, was a slim and good-looking girl when she was married, and throughout her life was endowed with more than a modicum of common sense. The mother of seven children, she complained of getting fat as she grew older. In 1723 she developed an umbilical hernia after the birth of her sixth child, but kept the fact to herself so strictly that the King did not know of its existence for many years. It increased steadily in size, and at last became strangulated, causing her death fourteen years after its first appearance. Lord Hervey in his memoirs gives a lively account of the final scenes, and as he was a favourite in constant attendance at Court his story is probably accurate in every particular. He says :—

"On Wednesday, the 9th. November (1737) the Queen was taken ill in the morning at her new library in St. James' Park. She called her complaint the colic, her stomach and bowels giving her great pain. She came home took Daffy's Elixir by Dr. Tesier, the German, and house-physician's advice but was in such great pain and so uneasy with frequent retchings to vomit, that she went into bed. However when the clock struck two and the King proposed sending Lord Grantham, to dismiss the company and declare there would be no drawing room, she, according to the custom of the family, not caring to own or at least to have it generally known how ill she was, told the King she was much better, that she would get up and see the company as usual. As soon as she came into the drawing room she came up to Lord Hervey and said ; 'Is it not intolerable at my age to be plagued with a new distemper ? Here is that nasty colic that I had at Hampton Court come again.' The Queen twice this summer at Hampton Court had been seized with a vomiting and purging which had lasted in the most violent manner for three or four hours and then left her so easy and well that she had played the same night in the drawing-room as usual and had talked almost with the same cheerfulness. This made Lord Hervey less alarmed than he otherwise

would have been for she looked extremely ill.   Lord Hervey asked her what she had taken and when she told him, he replied 'For God's sake, Madame go to your own room, what have you to do here.'   She then went and talked a little to the rest of the company and coming back to Lord Hervey, said, 'I am not able to entertain people.'   'Would to God the King would have done talking and release you,' replied Lord Hervey.   At last the King went away, telling the Queen as he went by that she had overlooked the Duchess of Norfolk.   The Queen made her excuse for having done so to the Duchess of Norfolk, the last person she ever spoke to in public and then retired, going immediately into bed, where she grew worse every moment.

" At seven o'clock when Lord Hervey returned to St. James's he went up to the Queen's apartment and found her in bed.   The Queen asked Lord Hervey what he used to take in his violent fits of the colic ; and Lord Hervey, imagining the Queen's pain to proceed from a goutish humour in her stomach that should be driven from that dangerous seat into her limbs, told her nothing ever gave him immediate relief but strong things.   To which the Queen replied ; 'Pshaw ! you think now like all the other fools that this is the pain of an old nasty stinking gout.'   But her vomitings, or rather her reachings, together with such acute pain continuing in a degree that she could not lie one moment quiet, she said about an hour after 'Give me what you will, I will take it.'   He fetched some snakeroot and brandy and asking Dr. Tesier who was in the outward room, whether he might venture to give it to her, Dr. Tesier, who was naturally timid and made more so by the manner in which he had been talked to in the King's illness last year, said the Queen's pulse was very high and feverish and as she was unused to drinking anything strong, he could not affirm that this very strong cordial would do her no hurt.   Lord Hervey then asked him if he should propose to the King to call in another physician, and if he had any objection to Broxholme ; and Dr. Tesier saying he wished it extremely but did not dare to propose it himself to the King.   Lord Hervey told Princess Caroline what had passed and would, if she approved, propose to the King that Dr. Broxholme might be called in. Dr. Broxholme was immediately sent for and when he came Tesier and he agreed to give the Queen some snake-root with Sir Walter Raleigh's cordial, but this cordial being long in preparing and Ranby, house-surgeon to the King, a sensible fellow, telling Lord Hervey that insisting on these occasions upon a cordial with this name or t'other name was mere quackery and that no cordial was better than another but in proportion to its strength some usquebagh was immediately given to the Queen who kept it about half an hour but then brought it up.   Soon after the snake-root and Sir Walter Raleigh's cordial arrived from the apothecary's. it was taken and thrown up about an hour after.   All these strong things had. without easing the Queen's pain, so increased her fever that the doctors ordered Ranby to bleed her twelve ounces immediately.   She took a glister but it came from her just as it went into her.   The King inconveniently both to himself and the Queen lay on the Queen's bed all night in his nightgown where he could not sleep nor she turn about easily.

" Early in the morning of Thursday the 10th. the Queen was blooded twelve ounces more on which her fever that had been very high all night.

QUEEN CAROLINE OF ANSPACH

abated and everybody but herself thought she was better. The vomiting was suspended for a few hours but nothing passed downwards and two glisters she took returned immediately and pure. This evening whilst the Princess Caroline and Lord Hervey were alone with the Queen, she complaining and they comforting, she often said 'I have an ill which nobody knows of' which they understood to mean nothing more than that she felt what she could not describe and more than anybody imagined. This night two more physicians were called in Sir Hans Sloane and Dr. Hulse who ordered blisters and aperients. These came up like every other thing soon after she had swallowed it and the blisters, though a remedy to which the King and Queen had often declared themselves very averse, were put upon her legs. At six o'clock on Friday morning the Queen was again blooded upon which her fever went almost entirely off; but the total stoppage and frequent vomitings still continued. The whole Friday the Queen grew worse almost every hour. The Princess Caroline went to bed at night in her own apartment; the Princess Emily sat up in the Queen's bedchamber; Lord Hervey lay on a couch in the next room and the King had his own bedding brought and laid upon the floor in the little room behind the Queen's dressing room and lay there until about four o'clock on Saturday morning when the Queen complaining more than ever of the racking pains she felt in her belly and of a throbbing, he was called and all the physicians immediately sent for.

" When the King came into the room to the Queen he whispered her, and, as he said afterward, told her he was afraid her illness proceeded from a thing he had promised never to speak of to her again; but that her life being in danger, he could not answer it to her, himself, or his family not to tell all he knew. She begged and entreated him with great earnestness that he would not and spoke with more warmth and peevishness than she showed at any other minute during her whole illness. However he sent for Ranby the surgeon and told him he apprehended the Queen had a rupture at her navel and bid him examine her. The Queen carried her desire to conceal this complaint so far that when Ranby came to feel her she laid his hand on the pit of her stomach and said all her pain was there; but Ranby slipping his hand lower, kept it there in spite of her some little time and then without saying one word to the Queen went and spoke softly to the King at the chimney upon which the Queen started up and sitting in her bed said to Ranby with great eagerness 'I am sure, you lying fool, you are telling the King I have a rupture.' 'I am so,' said Ranby, 'and there is no more time to be lost; Your Majesty has concealed it too long already; and I beg another surgeon may be called immediately.' The Queen made no answer but lay down again and turned her head to the other side, and as the King told me he thinks it was the only tear he saw her shed whilst she was ill. The King bid Ranby send immediately for old Busier the surgeon whom, though fourscore years old the King and Queen had a great opinion of and preferred to every other man of his profession. Busier not being immediately to be found and the King very impatient he bid go and bring the first surgeon of any note and credit he could find. When Ranby returned he brought one Shipton with him, a City surgeon and one of the most eminent and most able of the whole profession. By this time, too, Busier arrived and these three

attended her constantly. After they had examined the Queen, they all told the King she was in the utmost danger. Busier proposed the operation of cutting a hole in her navel wide enough to thrust the gut back into its place; which Ranby opposed, saying that all the guts upon such an operation would come out of the body into the bed, and that he thought he felt at the bottom of the swelling (which was of an immense size) a softness which he took to be a disposition to make matter and which they might encourage by warm fomentation till the swelling might break of itself or at least allow them by a slight touch of the lancet to open it without danger. Shipton inclining to Ranby's opinion, this method was pursued. About six o'clock this Saturday evening the surgeons lanced the swelling just at her navel and let out some matter but not enough to abate the swelling in any material degree or give them any great hope of her recovery.

"About four o'clock on Sunday morning the 13th. the Queen complaining that her wound was extremely painful and desiring to have it dressed, Ranby and Shipton were called in to her and upon opening the wound declared it had already begun to mortify. Hulse, whose turn it was that night to sit up, was sent for into the Queen's bed chamber and acquainted by the surgeons with the situation she was in. Hulse came to the Princess Caroline and told her this terrible and dreadful news upon which she bid him and Ranby go immediately and inform the King. When Hulse and Ranby came back to Princess Caroline (the King being already up and gone to the Queen) Princess Caroline and Lord Hervey asked Hulse if there was no possibility left of her recovery and he answered 'None.' Lord Hervey then asked Ranby if they were never deceived in the signs of a mortification, to which Ranby shaking his head, replied; 'We know them but too well.'

"The Queen finding the wound still so uneasy, sent again to have them open and dress it; but Hulse said it was to no purpose to do anything more and Ranby assured the Princess Caroline he could do nothing that would not give the Queen more pain, without a possibility of doing Her Majesty any good. However, the Queen insisting on having the wound cleaned at least, the King who had told the Queen all that the surgeons had told him, came out, called in Ranby and Hulse and made them comply with her request. As soon as the surgeons had applied some of their lenient ointments and anodyne preparations they left the room, came to Lord Hervey and confirmed their former report of the impossibility of her holding out many hours.

"She then took a ruby ring off her finger, which the King had given her at her coronation and, putting it upon his said; 'This is the last thing I have to give you—naked I came to you and naked I go from you. I had everything I ever possessed from you and to you whatever I have I return.'

"It is not necessary to examine whether the Queen's reasoning was good or bad in wishing the King, in case she died, should marry again. It is certain she did wish it, had often said so when he was present and when he was not present and when she was in health, and gave it now as her advice to him when she was dying; upon which his sobs began to rise and his tears to fall with double vehemence. Whilst in the midst of this passion, wiping his eyes and sobbing between every word with much ado he got out this answer; 'Non—j'aurai—des—maîtresses.' To which the Queen made no

other reply than ; 'Ah ! mon Dieu ! cela n'empêche pas.' I know this episode will hardly be credited but it is literally true. When she had finished all she had to say she said she fancied she could sleep. The King said many kind things to her and kissed her face and her hands a hundred times but even at this time, on her asking for her watch, which hung by the chimney in order to give it him to take care of her seal, the natural brusquerie of his temper, even in these moments, broke out which showed how addicted he was to snapping without being angry and that he was often capable of using those worst whom he loved best. For on this proposal of giving him the watch to take care of the seal with the Queen's arms, in the midst of sobs and tears he raised and quickened his voice and said 'Ah ; my God ! let it alone ; the Queen has always such strange fancies. Who should meddle with your seal ? Is it not as safe as in my pocket ?'

" The Queen after this fell into a sort of dozing, during which time the King often said ; 'She is dying ; she will go away in this sleep ; it is over ; she will suffer no more.' However when she waked she said she found herself refreshed and much better, adding ; 'I know it is only a reprieve to make me suffer longer, and therefore, I wish it was at an end ; for I cannot recover ; but my nasty heart will not break yet.'"

(To be continued.)

THE OPERATION FOR CLEFT PALATE.

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THE sufferer from cleft palate has one great disability ; he is unable to shut off the nose from the mouth at will. The object of operation must be the production of this missing power, and it is a reasonable assumption that the simplest way to produce it is to imitate the normal mechanism. Consequently the first step in planning the operation must be to define the exact manner in which the closing of the nasopharynx is normally performed. It is surprising, however, to see how little attention is given to this point in standard works on cleft palate. Usually the operation is treated almost as if it resembled that for hare-lip in having for its object appearance rather than function. In spite of the soft palate's being composed of actively contractile muscle, it seems to be assumed that it closes the nasopharyngeal passage by falling passively against the posterior wall of the pharynx, in the manner of a flap valve. Arbuthnot Lane, for example, amid almost universal approval, taught an operation which consisted in forming a soft palate simply of two layers of mucosa, not at all unlike a cardiac valve in structure. Similarly, in books of anatomy and physiology the action of the muscles of the palate and the nasopharynx is described in the vaguest and briefest way, with no attempt to show what actual use to the body their contractions serve. In this paper an attempt is made to give an admittedly incomplete account of the action of the normal soft palate, and from this and the principles of general surgery to deduce a technique of operation. Whatever originality emerges in this process is mainly in the arrangement of well-known facts, and in the conclusions drawn from them.

MECHANISM OF THE SHUTTING OFF OF THE NOSE FROM THE MOUTH.

I am convinced that the mechanism which closes the nasopharynx is the same as that which closes so many other passages in the body—the device of a complete muscular ring or sphincter (*Fig.* 1). This ring is set in a plane running backwards and slightly upwards from the middle of the soft palate to the posterior pharyngeal wall, just below the adenoid bed, at the level of the foramen magnum. It is not a simple circlet like the sphincters of the bowel, but is composed of many bands of muscle, interlacing and decussating with each other in the manner familiar to anyone who has dissected this region.

The complicated formation of this sphincter can best be understood if it is separated into anterior and posterior halves (*Fig.* 2). Each half consists

of a muscular sling, and each sling consists of two pairs of muscles, interacting with each other in curious ways.

The posterior sling is made up of a specialized band of the superior constrictor, and of the fibres of the palatopharyngeus which spread out over it. These two muscles by a simultaneous contraction produce a

FIG. 1.—Median section of the head, showing the way in which the nasopharyngeal sphincter shuts off the nose from the mouth in saying 'Ah.' Passavant's ridge is projecting forward to meet the posterior surface of the soft palate where it is dragged back by the levatores palati. The uvula is curled forward, and the posterior edge of the palate is not in contact with the back wall of the pharynx.

shelf on the posterior wall of the pharynx which is well known as Passavant's ridge.[8] though of course it is simply the posterior half of the septum produced by the action of the sphincter. The formation of this ridge is peculiar, as it is partly due to the active contraction of the section of the superior constrictor which forms it, and partly to the

FIG. 2. Diagrammatic drawing of the base of the skull, showing the muscles forming the sphincter (in solid black). This figure is the foundation for the series of diagrams in *Fig. 6.* It shows the formation of the two slings, and omits the palatopharyngeus as complicating the picture. There is, of course, a certain amount of distortion in order to force a scheme of three dimensions into the limits of two: but if anyone can design a better diagram I shall be only too willing to adopt it.
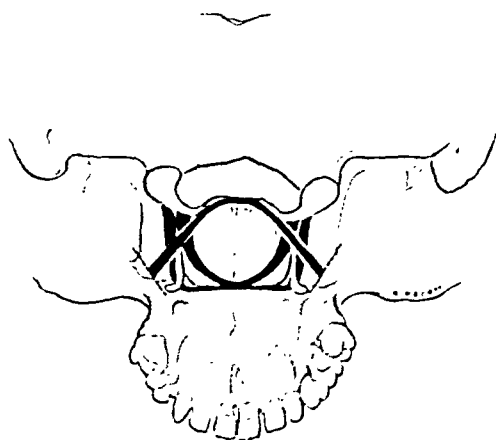
passive folding of this section into a ruck, by the lifting action of the palatopharyngei (*Fig. 3*). Their action can be illustrated by lifting a trouser leg, representing the constrictor,

by the two fingers and thumbs on either side, representing the palato-pharyngei. As the cloth is pulled upwards it falls into a ruck, but in the case of the pharynx this ruck itself contracts actively at the same time as it is lifted.

The anterior sling consists of the two levatores and the two tensores of the palate. The action of the levatores is the obvious one of lifting and dragging back the centre of the palate against the posterior half of the sphincter; but the tensores, instead of helping this action as the palato-pharyngeus helps the action of the superior constrictor, act in definite opposition to it. This is because of the change in direction given to their tendons by their turn round the hamulus of the pterygoid, so that, though the bellies of tensors and levators are so closely alike in origin and direction, the tensor pulls the palate down against the upward pull of the levator. The result is
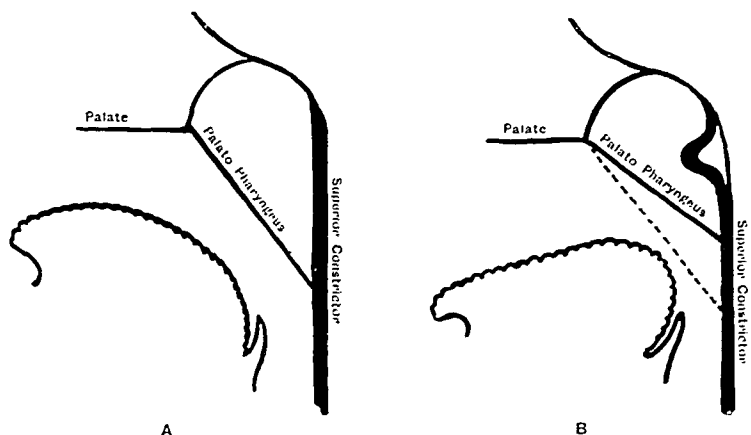
Fig. 3.—Diagram of the formation of Passavant's ridge. A, The posterior sling relaxed, with the constrictor flat against the vertebræ behind, and the palatopharyngeus at full length; B, The way in which the contraction of the palatopharyngeus pulls up the constrictor into a ruck on the posterior pharyngeal wall.

the same as with any other opposing pair of muscles in the body, for instance, the biceps and triceps of the arm : the structure under their control can be kept rigid in any desired position. The importance of this for voice production is obvious. It is interesting to note that this theory of the action of the muscles is borne out by their nerve-supply. The tensores palati are supplied by the otic ganglion, while the muscles which blend into the sphincter are all innervated by the pharyngeal plexus.

Once the sphincter thus described is recognized, it becomes obvious. It can be seen in action by a throat mirror, or felt to grip the examining finger like the sphincter ani. One of the best demonstrations of its action is to push a small rubber catheter through the nose down into the throat, and with this in position to swallow. As the sphincter contracts it rises, owing to the action of the palatopharyngeus already described, and the consequence is that the catheter is gripped and forced out of the nose for a short distance every time the nose is shut off from the mouth in the act of swallowing.

In speech the action of the sphincter is much lighter than in swallowing or gagging, but if the lower end of the catheter be watched through the mouth during the saying of ' Ah ', it will be seen to move upwards and towards the centre line. The mechanism is the same in both instances, but the degree of action is very different. Compare the difference between the light touch of the lips in talking, and their forcible screwing up to prevent the entrance of a tongue-depressor.

It is curious, however, to see the disbelief with which a diagrammatic drawing of the sphincter (see Fig. 1) is greeted by those used to the old conception of the action of the palate. And yet it merely consists of the co-ordination of two easily observed actions of the body. The first of these is the formation of Passavant's ridge, which is best seen when a cleft-palate case gags and the constrictor sling leaps forwards at each spasm (Fig. 4). The second is the action of the palate when the nose is shut off from the open mouth in saying ' Ah.' It will be seen that the middle of the palate goes back into a sort of dimple with the contraction of the levatores, while the posterior edge does not touch the back of the pharynx, as it should if it were acting as a flap valve. On the contrary, the azygos uvulae, acting in concert with the other muscles of the same innervation, curls the posterior edge of the palate forwards. Thus it is not necessary for the proper action of the soft palate that it should be long enough for its edge to reach the posterior wall of the pharynx, as is so often stated. Perfect control of the nasopharyngeal opening, and consequently perfect speech, is possessed by many in whom, owing to congenital defects, or to surgical attacks on the tonsil and uvula, the palate is so short that its edge can never meet the back wall.
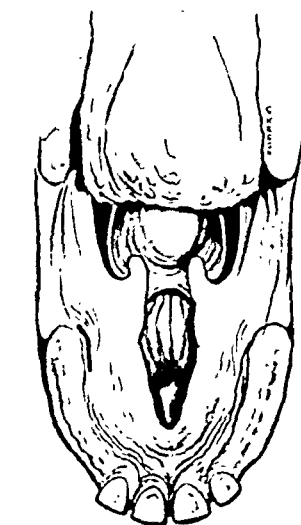
Fig. 4.—Drawing of the preliminary operation showing the appearance of Passavant's ridge when the patient is gagging after the removal of the tonsils, whose fossæ are seen to be empty. The incision for dividing the posterior palatine artery is shown on the left.

If the foregoing account of the action of the soft palate is accepted, the aim of the operation obviously becomes the construction of a contractile ring, as near as possible in structure to the normal one, and capable of closing the nasopharyngeal passage. In order to be effective this sphincter must be continuous in front with an air-tight septum between the nose and the mouth. In clefts involving the soft palate alone this septum is of course the natural hard palate, but in complete clefts an artificial junction of the sides has to be accomplished in this region too.

The conception of a sphincter as the final result of the operation at once eliminates flap operations of the Davies-Colley and Lane variety. It also explains their notorious failure to give normal speech by means of the thin layers of epithelium and scar tissue which they produce. Similarly, the recollection of the proper function of the anterior sling should

prevent a thin atrophic soft palate, containing hardly any muscle (*see Fig.* 6 *e*) from being called, as it so often is, a 'perfect anatomical result' of operation.

SOME ANATOMICAL CONSIDERATIONS.

It is, of course, impossible to give a formal description of the anatomy of cleft palate within the compass of this article. The following observations merely emphasize certain points which have a practical bearing on the performance and results of the operation.

1. **Increase of Surface and Decrease of Tissues in the Deformed Parts.**— The normal palate, like other parts of the body, has a definite proportion between the area of its epithelial surface and the mass of the tissues beneath. In cleft palate, as in the similar condition of hare-lip, there is a most important change in this proportion ; the area of the epithelium is increased and the mass of the tissues is diminished. The surface is increased because the rudimentary epithelium covering the edges of the palatine processes, which should have been absorbed when they met and fused in early fœtal life to form the palate, has survived to develop into normal membrane. The tissues beneath, on the other hand, are lacking in bulk, partly from original failure to develop, and partly from the lack of that stimulus to growth which only use can give. This lack of bulk is for some reason dogmatically denied by certain writers, but there is never enough tissue present to make a palate of normal fullness, even though one of normal function can usually be produced.

The aim of operation has already been defined as the creation of a soft palate as nearly as possible resembling the normal one in structure, and consequently in function. One of the most important elements in the resemblance is the proportion between the surface area and the mass of tissues it covers. In consequence it is not only permissible, but it is imperative, to remove large amounts of mucosa from the surfaces to be joined. The omission to do this is one of the commonest causes of a result like that in *Fig.* 6 *e*; in which the soft palate consists mainly of mucosa which should have been removed at operation.

This disproportion is, of course, the exact opposite of that which is found in plastic operation for injuries. In these the problem is to restore the loss of epithelium from a normal or nearly normal amount of tissues beneath. The distinction is a very important one, and failure to draw it has been responsible for a good deal of loose reasoning. In cleft palate, as in hare-lip, to preserve every scrap of surface is to ensure a bad result.

2. **Formation of the Bony Boundaries of the Cleft.**—It is strenuously maintained by Brophy that in cleft palate there is invariably a widening of the alveolar arch beyond the normal. A widening certainly exists at birth in some cases, though not by any means in all. Even without operation this widening disappears as the child grows, and apparently no adult skulls can be found to show it. The widest clefts of the palate are associated with wide clefts of the lip, and the joining of the lip in infancy has a very marked effect in narrowing the alveolar arch. Brophy's belief justified his well-known method of pulling the alveolar ridges together in the first months of life, but

apart from many other objections it seems to me that the sacrifice of the germs of the permanent teeth by leaving septic wires among them for long periods is too high a price to pay for an easier joining of the palate.

3. Blood- and Nerve-supply of the Palate. — The blood-vessels and nerves of the palate can be described together in an unsystematic but useful manner as falling into three main groups.

The first group is the posterior one, supplying the soft palate. The blood-vessels in it are several small branches from the external carotid. Its nerves are the motor branches to the muscles from the otic ganglion and the pharyngeal plexus, and the sensory twigs to the mucosa from the glosso-pharyngeal nerve. From the operator's point of view the most important fact about this group is that none of its constituents is in any danger of injury in the procedures recommended in this paper.

Secondly there is the middle group. This consists of the posterior palatine artery and the sensory nerves to the hard palate which accompany it. In contrast to the posterior structures, these cannot be ignored in the operation. They tether down the mucoperiosteum of the hard palate, just at the point where most mobility is required, by their emergence from the bone at the posterior palatine foramen.

The posterior and the middle group remain the same whatever the degree of deformity, but the third group, consisting of the branches of the nasopalatine artery and nerve which enter through the incisive foramen, varies according to whether the cleft involves their point of entry or not. If the septum is separated from the hard palate by a complete cleft, it is of course impossible for either artery or nerve to reach it. This is, however, of no practical importance ; the anterior end of the posterior palatine artery must always have some anastomosis with a similar terminal twig of one of the many branches of the internal maxillary, which give this region so rich a blood-supply. In consequence there is always available an anterior source for the collateral circulation to be described later.

4. The Point of Danger.—It is well known that breakdown of the line of junction after operation almost invariably starts at the junction of the hard and soft palates. The reasons for this being the point of danger lie in the anatomy of the parts, and may be described under four heads :—

a. The clefts that are the most difficult to close (the complete or nearly complete ones) are at their widest at this point. In front the sides always slope together till the alveolar ridge is reached, and posteriorly the halves of the uvula can always be made to meet with ease.

b. There is in this region a sudden change in the substance and shape of the tissues to be joined. The thin, tough, and immobile mucoperiosteum of the hard palate leads without graduation of any sort into the thick, soft, and actively contractile muscle behind. This change calls for different methods of joining the different tissues, and for special care at the point of transition.

c. The three strongest anchorages of the flaps to be joined are all concen-trated here. The actual line of junction between the hard and soft palates is marked by the insertion of the palatine aponeurosis into the posterior edge

of the bony palate. In front of this there is the posterior palatine artery, and behind it the tendon of the tensor palati.

d. As the muscles of the soft palate pull backwards as well as upwards on the line of healing, any split due to their action will begin at the anterior border of the muscular ring rather than at the posterior one.

5. **The Line of Division between the Muscles of the Sphincter and those External to it.**—This line of division is the pterygo-mandibular raphé, which has the sling of the superior constrictor arising from it on its inner and posterior side, and the buccinator taking origin from it anteriorly and externally. As might be expected from analogy with other regions of the body, no important vessels or nerves cross this raphé, and an incision along its line to detach the sphincter from the structures to its outer side can be made with a minimum of damage.

SOME GENERAL PRINCIPLES OF OPERATION.

The tissues to be moved, and the functions to be performed by them in their final position, differ so widely in the two divisions of the palate that these divisions are best discussed separately. In the hard palate the sole object is the making of a septum, much as in an operation for the cure of a vesico-vaginal fistula. In the soft palate, however, there is the very different problem of moving muscles in order to give them a definite function in their new position, and many of the rules governing this type of work elsewhere have to be observed. It must never be forgotten that the production of a simple stiff partition between the nose and the mouth, which means success in dealing with the hard palate, means failure when dealing with the soft.

The Hard Palate.—

1. *The Closing of the Gap.*—The mucoperiosteum of the hard palate is practically unstretchable, so that it must be shifted in one way or another to cover the central cleft. In the classical Langenbeck operation the mucoperiosteum on either side is detached from the underlying bone to form two flaps, attached along their outer margins to the alveolar ridges. These flaps are then swung downwards till their inner margins meet in the mid-line like the sides of a double door. The amount of freedom gained by this swinging on the hinge of their attachment to the alveolar margin is strictly limited by the width of the flap and the inclination of the palatine process on which it lay, so that in many cases, especially those with flat arches to the roof of the mouth, it is insufficient to give easy joining in the mid-line (*Fig. 5 b*).

If, on the other hand, the mucoperiosteum is boldly detached from the alveolar ridge along its outer border so that it is simply left attached by its anterior and posterior ends, it can be pulled inwards to any extent needed by the width of the cleft, and still left in contact with the underlying bone. For reasons to be given later this keeping of contact with the bone is of great importance (*Fig. 5 d*).

2. *The Gaining of a Wide Area of Contact between the Flaps.*—It is obvious that the wider the area of contact between the raw surfaces of the flaps, the better the chance they have of joining together rapidly and firmly. The simple edge-to-edge junction of the Langenbeck method has a comparatively
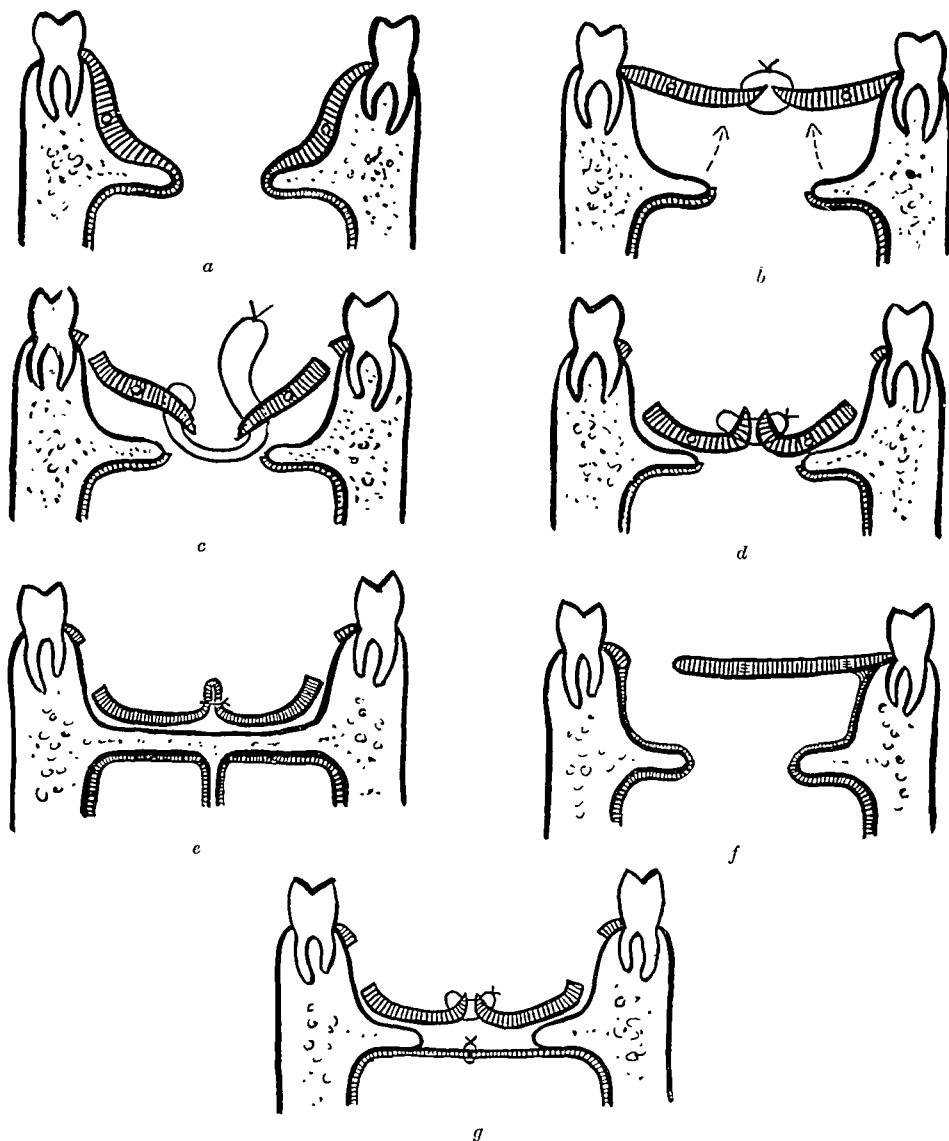
Fig. 5.—Diagram showing the treatment of the hard palate. (a) Untouched cleft of hard palate. The septum is ignored for the sake of simplicity. (b) Cleft closed by Langenbeck's method. The mucoperiosteum has been swung out from the bone on the hinge of its attachment to the alveolar ridge, till the edges of the cleft meet. The upper surfaces of the mucoperiosteal flaps are left bare, to granulate and contract. (c) Recommended method of treating the mucoperiosteum. It has been detached from the alveolar ridge as well as from the palatal process, and a vertical mattress stitch has been inserted. (d) Result of tightening the suture inserted in the previous diagram. The edges of the cleft have been everted against each other, and the mucoperiosteum will heal firmly to the bone in its new position. The raw surfaces left laterally are of no importance, as they have a floor of bone. The dangerous area of raw surface is confined to the actual width of the cleft in the bone. (e) Method of raising a ruck in the thin mucosa above the apex of an incomplete cleft. (f) Formation of a lateral fistula, owing to insufficient mobilization of the flaps, which has not allowed the joined mucoperiosteum to settle against the bone. (g) Diagram to show the mucosa lining the floor of the nose joined by Veau's method, as an addition to the closure of the roof of the mouth shown in (d).

poor expectation of junction; but if a strip on either side at least 5 mm. wide can be everted against its opposite flap, healing is almost certain. This eversion can be quite easily obtained by means of a vertical mattress stitch, provided that the flaps are sufficiently freed.

When the cleft does not extend throughout the hard palate, it is necessary, in order to gain this eversion at its apex, to insert a couple of mattress sutures in the intact mucoperiosteum in front of it (*see Fig. 7*) in order to raise a ruck there in the mid-line. This means that instead of having an edge-to-edge contact of the thin tissues that normally form the apex of the cleft, there is a wide junction. It is a simple trick, but an almost infallible guarantee of success in the treatment of an incomplete cleft (*Fig. 5 c*).

3. *The Maintenance of the Blood-supply to the Healing Surfaces.*— Healing in any part part of the body varies in certainty and speed with the abundance of the blood-supply. It has already been stated that free mobility of the lateral flaps is essential, and also that the posterior end of the muco-periosteum of the hard palate is tethered to the bone by the structures emerging from the posterior palatine foramen. If these are left intact, they anchor the flaps at just the point where mobility is invariably most needed; yet if they are cut at the time of operation, there is both free bleeding from the severed artery, and, what is more important still, a great diminution of blood to the healing surfaces. The solution of this dilemma appears to me to be the deliberate arrangement of an adventitious circulation to replace the natural one, by the cutting of the posterior palatine artery at a preliminary operation. This means the destruction of the middle artery of supply to the palate, and the taking over of its functions by the natural enlargement of its anastomoses at either end. It is a manœuvre strictly in accordance with the changes of circulation deliberately arranged in plastic surgery elsewhere—for instance, in caterpillar flaps.

The unavoidable cutting of the sensory nerves at the same time as the artery I regard as of no real importance. They must regenerate in time in most cases, and I have never seen the slightest trouble from their section.

4. *The Removal of the Strain of Junction from the Soft Palate.*—However free the mobilization of the sides of the cleft may be, there must remain an irreducible minimum of tension to be overcome in holding them together. This tension should be put as much as possible upon the tough and unimportant tissues of the hard palate, rather than upon those flimsy muscles of the soft palate which determine normal speech. If the hard palate is dealt with in the way recommended, it will be found that the sides of the gap in the soft palate are almost or quite in contact, and need only light co-aptation sutures to hold them together.

5. *The Minimizing of Post-operative Contracture.*—Wherever an area of the body is left denuded of epithelium, that area tends in the process of healing to contract forcibly. It is contraction of this type which causes the narrowing of the alveolar arch which is so common after the joining of a cleft of the hard palate, and which also fixes and stiffens the soft palate.

In the Langenbeck operation the raw area left is immense—the entire width of the alveolar arch in many cases—and there has been very little discussion as to how exactly this heals. Raw areas must be left in any

conceivable operation, but there is a very important distinction to be drawn between two different types of these. In the first the raw area is an exposed surface of bone, the rigidity of which is quite capable of resisting the contracting force exercised upon it in the process of healing. The gaps left at the outer edges of the hard palate flaps are of this sort, and in consequence cause no contracture and are entirely harmless. In the second type the raw surface is unsupported by bone, and exerts its full pull upon the structures at its margins. It is this kind of healing area which is left upon the upper surface of the mucoperiosteum after Langenbeck's operation, and its contraction bends the bony arch above as a bow-string bends a bow. In addition to this action it must drag the upper surface of the soft palate backwards and downwards, fixing it in the worst possible position for fulfilling its function. If the hard palate flaps are slid inwards in the way recommended, the raw surface of the dangerous type left is no more than the area of the actual bony gap, instead of extending from one alveolar ridge to the other. By adapting the procedure of Veau (*Fig. 5 g*), and making a complete lining to the floor of the nose from the mucosa of the palatal process and the vomer, it should be possible to reduce this dangerous raw area almost to nothing. I have had insufficient experience of Veau's technique to judge whether the making of this inner nasal lining is worth while, but in complete clefts it seems a most reasonable proceeding, and there is no great difficulty in adding it to the joining of the oral surface.

The lateral fistula that is sometimes described as persisting after the making of relaxation incisions in the hard palate is the result of half measures —that is to say, the freeing of the flaps has only been enough to let their edges meet in the middle, and not enough to allow them to fall against the bone (*Fig. 5 f*). In consequence the inner side of the lateral incision is held stretched away from the bone till epithelium grows over it in this position. If the flaps are properly mobilized this cannot occur.

The Soft Palate.—

1. *The Closing of the Gap.*—The simplest method of closure, that of simply sewing together raw surfaces produced on either edge of the cleft, fails in such a large number of cases that many procedures have been devised for combating the tension which is the most obvious cause of breakdown.

Fergusson,[5] in a pre-anæsthetic and pre-antiseptic era, obtained a remarkable percentage of joins, in incomplete clefts, by cutting the actual palate muscles on either side. It is hardly necessary to point out the disadvantages of this Gordian solution of the problem.

Arbuthnot Lane[7] ignored the muscles completely, and closed the gap with a flap of mucosa. If voluntary closure was ever possible after this, it could only be by an abnormal development of the posterior sling.

Brophy[4] and Veau[9] rely on secondary tension sutures of wire which stretch the muscles to such an extent and for such a time that healing can occur in the mid-line. The pressure and the inevitable sepsis must of course cause considerable scarring and necrosis in the very tissues on which speech depends, but both surgeons recommend these sutures as avoiding the damage done by relaxation incisions (*Fig. 6 d*).

Gillies,[5] with a welcome recognition of the importance of function rather than appearance, has suggested detaching the soft palate entirely from the hard, and suturing it separately. However, as perfect function can admittedly often be attained combined with complete closure, it seems a counsel
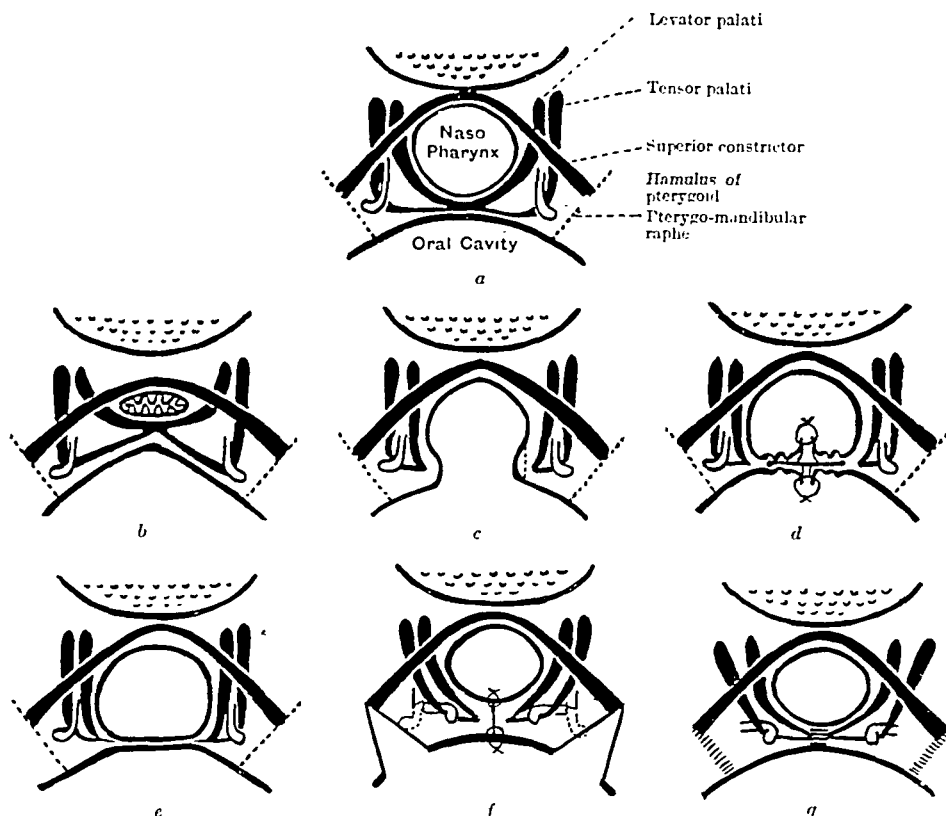
FIG. 6.—Diagram showing the treatment of the soft palate (*see Fig. 2*). (*a*) Normal sphincter in relaxation. The constrictor and the levator palati are relaxed, and the tensor is contracted, pulling the soft palate away from the posterior pharyngeal wall. (*b*) Normal sphincter in contraction. The tensor is relaxed, allowing the middle of the soft palate to go up into a dimple. The constrictor and the levator are contracted, shutting the nasopharyngeal passage. (*c*) Formation of a cleft of the soft palate. The bony framework is the same as before, but both the muscles of the anterior sling are shortened, and end not far from the hamular process. The line of section of the mucosa advised is shown on the right. (*d*) Method of joining palate by splitting the edges of the cleft and suturing them, while the strain is taken by a submucous suture of the type of Veau's. The results of other types of relaxation sutures can be easily substituted. (*e*) The result usually produced by suturing without shifting the muscles. The palate is thin and tight, and rather farther away from the posterior wall than normal. (*f*) Method of suturing recommended. Relaxation incisions have been made along the pterygo-mandibular raphé, and the hamulus has been snapped off inwards. This allows the ends of the muscles to meet without stretching. A wide strip of mucosa has been removed, and the raw surfaces are joined by two rows of sutures. (*g*) Result aimed at by the operation recommended. The nasopharyngeal passage is narrowed, the ends of the muscles are firmly joined, and the relaxation incisions have healed into a reinforcement of the pterygo-mandibular raphé.

of despair to advocate the payment of the very heavy price of a permanent gap in the palate for the chance of improved speech. I believe that the method recommended in this paper gives even better freeing of the muscles,

combined with better prospects of healing ; and it does not condemn the patient to prosthetic appliances that are both difficult to fit and galling to wear.

Finally, the usual text-book of general surgery vaguely advises that " relaxation incisions should be made when closure cannot be otherwise effected ", and in its illustrations shows these incisions so placed that their only effect could be to produce dangerous bleeding from the posterior palatine vessels.

It is curious that almost the only solution of the problem that is not advised is to apply to the palate muscles those principles which would be used if they were situated elsewhere in the body. The operation is essentially one of muscle transplantation. The well-known rules of this proceeding are to leave the muscle untouched at its origin, and to free it distally enough to allow an easy reaching of its new insertion, without undue tension. As the purpose of the shifting of the palate muscles is the closing of the gap in a sphincter, this insertion is for each muscle the end of its corresponding one on the other side.

The rigid bony framework of the pharynx is of normal size ; the muscles available are short and atrophic. Consequently they need considerable loosening to give an unstrained line of junction, just as the facial muscles need loosening from the underlying framework of the face in closing a cleft lip. The freeing of the levator palati is easy enough, but the turn of the tendon of the tensor palati round the hamular process fixes it firmly to the boundaries of the nasopharynx, as well as changing its direction so that it pulls directly outwards against the line of junction. It is fortunate that the hamular process can be very easily snapped off at its base without interfering with the synovial sheath of the pulley, and so allowed to be displaced inwards and upwards to a position which will not interfere with the joining of the two tensores. In this new position it must finally become fixed by the healing processes so as to afford once more a fulcrum to the tendon that curls round it (*Fig. 6f* and *g*).

2. *The Neutralization of Muscular Pull upon the Sutures.*—In the previous section the problem of tension has been regarded as if the tissues concerned were passive structures, whose pull upon the healing line would vary simply with their elasticity and the thoroughness with which they had been mobilized. But there is another kind of tension to be considered as well, the more dangerous and variable active tension due to the contractions of the irritated muscles. These contractions, however, can be robbed of all their danger if the process of joining the gap leaves the distal end of the muscle at a distance from its origin less than its minimum length when contracted. The mobilization recommended does this, and in time the muscles shorten to suit the new conditions, just as the muscles of a limb do when their supporting bone loses length from a fracture.

3. *The Diminution of the Diameter of the Passage to be Controlled by the Sphincter.*—This is another secondary, but very important, effect of the shifting of the palate muscles. It is obvious that if tissues of less than normal length are to be joined into a ring at no more than normal tension, that ring will be proportionately smaller than the normal diameter. *Fig. 6f* shows

how the falling upwards and backwards of the line of junction of the gap in the sphincter diminishes the diameter of the passage it has to control, bringing the front half of the sphincter close to the back half (Passavant's ridge). The importance of this in facilitating voluntary closure is easy to understand.

4. *The Provision of a Broad Healing Surface.*—This is as important in the soft palate as in the hard. The best way to gain it is by free removal of mucosa, which, as has already been pointed out, is present in excess. The method so often recommended, of splitting the edges of the sides, and suturing the split halves separately has three disadvantages. First, there is a central dead space left, in which blood-clot can accumulate and nourish infection; secondly, healing will produce a median strip in the soft palate consisting of mucosa alone, instead of a proper junction of the actual muscles; and lastly, the preservation of all the mucosa means that there is a surplus available to line any gap that may appear in the line of suturing, with a consequently greatly decreased tendency of this gap to spontaneous closure.

5. *The Maintenance of the Blood-supply to the Healing Surfaces.*—There is fortunately no danger of cutting the main arteries of supply to the soft palate, as has been already pointed out. It is, however, easy to produce local areas of anæmia by sutures and other devices used to counteract tension, and these areas must suffer from reduced healing power. Fergusson, for instance, who used a suture embracing the entire thickness of the palate, notes that often the only places that failed to join were those directly opposite the stitches. The moral is to reduce this interference to a minimum by employing two layers of fine sutures on the oral and nasal surfaces respectively, which leave between them a broad healing area with a completely free blood-supply. If the mobilization is sufficient there is no need for tension sutures, tapes, and metal devices, which must interfere gravely with the supplying vessels, besides destroying important tissues.

6. *The Minimizing of Post-operative Contracture.*—The comparatively large amount of scar tissue produced at the outer edge of the sphincter by the free mobilization of the muscles does much less damage than a smaller amount in more vital spots. The main mass along the lateral incisions simply reinforces an already existent line of fibrous tissue, the pterygo-mandibular raphé.

As the lateral incisions heal they have a tendency to drag back the displaced tissues into their original positions, but in assessing this tendency it must be remembered that the central suture line has the advantage of healing first, and that the new position of the muscles is confirmed by every contraction they make.

PRELIMINARY OPERATION.

There are two things to be done as a preliminary to the actual joining of the cleft :—

1. The first of these is the rearrangement of the blood-supply by cutting the posterior palatine artery. It is easily done by making a small incision just to the outer side of its emergence from the bone, and then tearing it

through with a dissector. The division of the artery is signalled by a violent gush of blood, which soon ceases under pressure on the wound.

2. The second preliminary is the removal of that forcing bed of infection, the tonsil. I am aware that some followers of Pangloss[2] consider that this organ has a protective function, but personally I believe it belongs to the same order of nuisances as the mastoid cavity, and has as much function as the male breast. It is one of those waste places and weak spots which pervade the human body as they do the human mind. I follow Addison[1] in thinking that the risk of that violent infection of the wound which is the only cause of failure in a properly performed operation is much decreased by previously removing the tonsils. They should be dissected out with the utmost care to preserve every scrap of mucous membrane ; there is no excess of surface where they lie. In two of my most difficult cases the guillotine had been used, with the not uncommon result of removing equal proportions of tonsil and soft palate. I do not think the adenoids are ever so dangerous as the tonsils, but if they have accumulations between their folds or appear inflamed they should be removed.

At least three months should be allowed after the preliminary operation for the resolution of all inflammatory products before the main operation is performed.

MAIN OPERATION.

Naturally the child should be free from colds and in as good general health as possible. There is much to be said, in London at any rate, for considering it an operation of the summer season.

I think that the liability to infection of the wounds is undoubtedly diminished by the administration of a prophylactic dose of 5 c.c. of anti-scarlet-fever serum a day or two before operation, as advised by Addison. There is a close connection between the more dangerous throat streptococci and the organism of scarlet fever.

Anæsthetic.—Intratracheal gas and oxygen through a nasal catheter is the best of all, but few are competent to give it. Ether vapour delivered through a weighted self-retaining tube is quite satisfactory. The very greatest care should be taken to keep the airway free and the colour good.

Position.—The patient should lie on a flat table, with the shoulders raised about 3 in. by a sand-bag. This position allows blood to run into the back of the nose rather than down the throat, but the head is not congested as it is if lowered below the main part of the body. The surgeon should sit at the top of the head ; thus he sees the structures upside down, as they are illustrated.

Instruments.—I do not think there is any need for the special instruments which fill so many pages of the instrument-makers' catalogues. The problem is that of stitching accurately in a cramped space, and there are few major operations in which it may not arise. The repair of a common bile-duct or of a ureter may expose the limitations of the ordinary needle and needle-holder just as well as the stitching of the nasal surface of the soft palate. Consequently the indication is not to invent special varieties.

of these instruments for cleft palates, but to improve the ones in ordinary use till they will pass this or any other test.

The needle-holder I use is one primarily designed for general use, but I have found it to add greatly to the ease of this operation (*Fig.* 7). It has a very wide range of movement for any given position of the hand, it is gripped equally firmly whether open or closed, and the catch can be released by a touch on the trigger without twisting the point, however it is held. To most people control of the jaws by the thumb and release of the catch by the forefinger come automatically.

Fig. 7.—Needle-holder and needle described in the text. The needle is more slender than it appears in the illustration.

The needle is 25 mm. in length. It is curved through five-eighths of a circle, so that the point can be inserted into a flat surface and made to re-appear after taking a full grip, simply by twisting it. If the curve of the needle is merely half a circle or less it is some-times difficult to get the point to show after inserting it in the deeper stitches. It has a sharp cutting edge, but this is confined to the extreme end, so that it cannot cut sideways if dragged askew. The ordinary cutting needle with a long edge on either side may do great damage in this way. No other needle is necessary in closing cleft palates, or for that matter in closing hare-lips. Like the needle-holder, it is an instrument of general surgery, with many applications in the under-running of bleeding points and the closure of minute rents. The holes made by it are punctures rather than cuts or tears, and so the tissues lie closely around the sutures, with a consequently diminished liability to infection compared with those that surround the gaps left by the harpoon-like instruments sold as 'cleft-palate needles'.

The sutures should be silkworm gut, which has no capillary attraction for the necessarily infected fluids of the mouth. Fairly thick strands should be used for the hard palate, and finer grades for the soft. There is no need to use the very finest available gut; apart from its tendency to break on tying, it must cut through the tissues more easily than the moderately thick.

An ordinary scalpel, an extremely sharp fine-pointed one, a curved 7-in. Mayo's scissors, a semi-sharp straight dissector, and a fine rat-toothed dissecting forceps complete the outfit needed for the actual operation. In addition a really powerful suction pump to clear away the blood will be found as great a help in this operation as it is in dealing with an appendix abscess or a brain tumour.

Mobilization of the Sides of the Cleft.—The anatomical and technical principles set out above remain the same whatever the extent of the deformity may be. It follows that the operation is much the same whether the cleft is wide or narrow, complete or incomplete. In every case without exception both divisions of the palate, the hard as well as the soft, should be completely
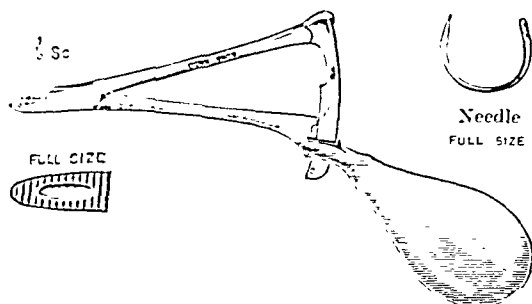
mobilized. This mobilization is obviously necessary when the cleft is complete, but it is also needed in incomplete clefts if the principles of getting broad healing surfaces in contact and allowing unimportant tissues to take the strain of suturing are to be followed.

The lateral incisions follow the lines described by Addison, dividing the mucosa along the whole length of the pterygo-mandibular raphé, and then running forward, close to the inner edges of the teeth, to about the level of the incisor (*Fig.* 8). The soft palate is freed from the buccinator and pterygoid muscles by blunt dissection along its outer side to a depth of a centimetre or more. On the inner wall of this incision the hamulus of the pterygoid can be plainly felt, just as it can be felt through the upper end of the gap left by removal of the tonsil, and it should be snapped off inwards. This can be done by a surprisingly light pressure with the finger nail. It should be remembered that the displacement of the spike of the hamulus leaves palpable its projecting base, which feels very like the original process. The hamulus is never exposed to view, and no damage is done to the synovial sheath of the tendon of the tensor palati as it turns round it. Consequently this muscle can act efficiently again as soon as the hamulus is fixed by adhesions in its new position.

FIG. 8.—Diagrammatic drawing of a complete single cleft. On the right side the line of incision is marked out: the hamulus, the tendon of the tensor, and the posterior palatine artery, are shown as though the mucosa were transparent. On the left side the palate, both hard and soft, has been freed ready for suturing. In the bare palate bone left by its inward shifting can be seen the stump of the artery, which has been cut at a previous operation, while the hamulus is broken off its base and has fallen inwards. Of course the tendon of the tensor palati should not be exposed as it is in the drawing.

Next the mucoperiosteum of the hard palate is levered off the bone by the dissector, avoiding the entrance of the branch of the naso-palatine artery at its anterior end. As the posterior palatine artery has been previously cut, there is no obstacle to the complete and rapid freeing of this flap. Along the margins of the cleft the dissector should be thrust out till it is free in the mid-line, aided if necessary by a touch with the knife.

The last stage in freeing the palate is to separate it by the curved scissors and the dissector from the posterior edge of the bony palate—a proceeding which is made easy by the previous division of the posterior palatine artery. The proof of proper mobilization 'is that the sides of the cleft tend to fall together, and can be pushed into contact with the very lightest pressure.

Bleeding during this part of the operation is usually free, but no vessel of any size should be divided, and the capillary oozing can be controlled by pressure. It is during this stage especially that a proper sucker will add so much to the ease of the work.

Suturing the Hard Palate.—If the hard palate is cleft, the edges must be dragged together by vertical mattress sutures (*Fig.* 9) of strong silkworm gut till at least 5 mm. of the raw surface on either side is in opposition. At the extreme anterior end of complete clefts it is usually impossible to evert the edges, but a small defect left here usually closes spontaneously.

The procedure is much the same if the hard palate is normal, or only partially cleft. Mattress sutures are used to raise in the middle line the ruck previously described. The making of this fold drags together the edges of the actual cleft behind it, and ensures that at its apex there shall be broad raw surfaces in contact instead of thin edges. I would repeat that if the hard palate is properly treated, the sides of the cleft in the soft part will be almost or quite in contact before any sutures are put in them.

Suturing the Soft Palate. —The handling of the flimsy edges of the cleft of the soft palate is much helped by passing a fine suture through the tip of each half of the uvula. The ends of these sutures are caught in artery

Fig. 9.—Diagrammatic drawing of the stitching of a cleft of the soft and half the hard palate. Both sides have been freely mobilized, and a ruck has been raised at the apex of the cleft by two mattress sutures. Two vertical mattress sutures evert the edges of the muco-periosteum, and the sutures on the nasal surface of the soft palate are shown in place. A single one of the six sutures which will join the oral surface has been inserted.

forceps, which are allowed to hang forwards or backwards as is most convenient, to act as steadying weights. Then with the fine-pointed knife a strip of mucosa is carefully sawn off the edge of the cleft, starting the cut by transfixion close to the steadying stitch. This strip should broaden from about 4 mm. near the uvula to about 8 mm. where it meets the anterior raw surface, past the belly of the sphincter. For the reasons given the raw area should err on the side of wideness rather than of narrowness, and the greatest care should be taken to see that all mucous membrane is cleanly removed from it.

Then the nasal surface of the soft palate should be joined by interrupted sutures. The first of these is the most important in the whole operation, and should join firmly and accurately the angles caused by the sudden swell of muscle from the thin mucoperiosteum of the hard palate. It should take

up about a third of the thickness of the raw surface, and the needle should be felt to penetrate the tough layer of the palatal aponeurosis. The succeeding half dozen stitches on this surface take gradually decreasing bites of the tissues, till finally the uvula is formed by fine sutures of the mucosa alone. The insertion of the later stitches will be helped by letting the sutures stretching the sides of the cleft fall forwards over the nose. When this row of sutures has been completed the line of junction should appear as a deep narrow cleft, with straight sides that are in contact for most of their depth. The oral surface is next joined by half a dozen stitches, any dried blood is cleared away, and the operation is complete. I think that the use of Whitehead's varnish or any similar preparation is a mistake; it cannot make the suture line waterproof, but tends to collect and confine the exudation.

After-treatment.—There is very little to be done after operation beyond following the very obvious indications of keeping the child as contented as possible, and getting it to take soft food and an abundance of glucose water. The latter will clean the wound to some extent, and I do not think that syringing the mouth is worth the trouble it almost invariably causes. The whitish accumulations, usually called sloughs, are simply the sodden state of what would be a scab on a dry external wound, and are no more harmful on the palate than in the tonsillar fossa after a tonsillectomy.

That bugbear of the older surgeons, the breaking down of the wound by the use of the child's tongue, never occurs in actual practice; though it would seem natural enough to expect the continual investigation of a totally new structure occurring in the roof of one's mouth. Similarly, I doubt very much whether there is any justification for the use of arm splints.

Really the only thing to fear after the operation described is sepsis of that corroding type which will break down any wound in which it occurs. The action of the palate muscles can put no strain on the suture line for the reasons described. Indeed, I have encouraged several of my more biddable patients to demonstrate the action of the sphincter by talking and saying 'Ah' during the first week after operation, with the stitches still in position. Incidentally it is an interesting psychological point that the use of the sphincter in saying 'Ah' is instinctive, even if it is so incomplete that it can never have shut off the nose.

The stitches on either surface of the soft palate will come away by themselves, but those in the tougher tissues of the hard palate will remain indefinitely if left alone. Consequently they should be removed about the tenth day.

Results.—The free mobilization, neutralization of muscular contraction, and light suturing which have been described give a high percentage of complete unions. In partial clefts failure should be very rare, while in the complete ones it is easy enough to get runs of a dozen successive cases with primary healing of both divisions of the palate.

I would prefer to postpone the discussion of the functional results till a larger number of my patients (most of whom have been operated upon at about 2 or 3 years old) have attained the age of willing co-operation. However, the freedom of movement of palates formed in this way, upon saying 'Ah' or gagging, promises very well.

SUMMARY.

1. The object of the operation is assumed to be the imitation of the normal mechanism of shutting off the nose from the mouth.

2. This mechanism is argued to be a sphincter.

3. In forming this sphincter the ordinary principles of muscle transplantation should be observed.

4. As the muscles are too short to meet without tension they have to be shifted.

5. This shifting allows them to meet. narrows the passage they will have to control. and renders their contractions innocuous to the healing line.

6. This shifting cannot properly be done without changing the blood-supply at a previous operation.

7. Certain forms of raw surface are harmful. and others harmless : the operation is planned accordingly.

8. The tonsil is a danger to the success of the operation. and should be previously removed.

9. The operation is described.

I find it difficult to acknowledge properly what I owe to the privilege of watching the work of my seniors at the Hospital for Sick Children. more particularly Mr. Addison and Mr. Barrington-Ward. As for the illustrations by Dr. C. A. Keogh. there lies in them. if nowhere else. a justification of this paper. Their quality can be appreciated either by comparing them with those which adorn more famous works. or by endeavouring to produce oneself something approaching their simplicity and accuracy.

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A GIANT GALL-STONE IMPACTED IN THE COLON AND CAUSING ACUTE OBSTRUCTION.

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IN February, 1931, I was asked by Dr. J. E. Basham, of this city, to see an old gentleman of 81 suffering from acute abdominal pain with vomiting. I had known this patient by sight for nearly forty years and had always recognized him as a stout, healthy-looking man of regular habits and addicted to walking. The family doctor gave me the following history :—

" I was called to attend the patient in February, 1930, for an attack of bronchitis with very troublesome cough. When almost recovered from this illness, he complained of 'pain in the side with a feeling of distension'. The pain was intermittent and there was slight tenderness beneath the left costal margin which did not increase on deep pressure. He was at that time very stout and difficult to examine, and nothing definite was made out. The patient was of a constipated nature, and for years had been accustomed to take a laxative of his own concocting, but in spite of his remedy complained that his bowels had not been moved for two days. After an aperient the discomfort was removed and there was no further disturbance at that time. In July he suffered from retention of urine due to enlarged prostate. The radical operation was carried out, and by the end of September the patient was nearly well again. In November I was again called to see him regarding the pain in the side. The tenderness was more marked, and the pain, whilst not severe, was worse than on the previous occasion ; it was again intermittent in character ; the bowels were not constipated, but the evacuations were very watery and pale in colour. There was no apparent distension of the upper abdomen, but on deep pressure over the tender area the pain was made worse. By means of senna and cascara the bowels were kept open, and gradually the attack subsided. I was certain at this time that he had a chronic intestinal obstruction at the junction of the transverse and descending colon and informed the patient, but he declined further investigation and would not even consent to be X-rayed. No doubt his refusal was due to the fact that he was just recovering from the long illness due to his prostate, and as the attack eased off he wanted to leave things alone for the time being. Early in February, 1931, the patient again suffered from an attack of bronchitis lasting ten days, and just as this was clearing away I was again called to see him on account of recurrence of the pain in the side, the attack resembling that of November, 1930. This was two days before I asked you to visit him. On a Sunday morning he appeared better, but that evening he was much worse, had vomited twice, and there had been no movement of the bowels or passage of flatus. I gave an enema of

¾ pint of hot water, soap, and olive oil with a long tube, with the object of reaching the area where I thought the obstruction was situated. After inserting about two to three feet of tube I felt the end strike against something very hard, and on gently pumping the syringe no fluid would go through. I withdrew the tube an inch or two and tried again : this time the solution flowed without obstruction and the rubber tube could then be passed farther than where it had previously struck the hard substance. The patient obtained instant relief, which continued until the next morning, when he became worse and I called you in."

When I saw this old gentleman he was in the throes of an attack of intestinal obstruction and looked very ill. The abdomen was much distended and the contracting cæcum could be both seen and felt. The diagnosis appeared obvious enough, and I had no doubt that he was suffering from a malignant growth about the splenic flexure with a superadded acute obstruction. With as little delay as possible he was removed to a private hospital. Using local anæsthesia I exposed and opened the cæcum, with immediate and complete relief (this was on Feb. 16). His general condition rapidly improved and he began to take nourishment. Once or twice he felt as though the bowels might move, but there was only a small evacuation and he passed no flatus by that route. After the distension went down, a large mass could be felt in the left hypochondrium and this was looked upon as undoubtedly a new growth in the large bowel. During succeeding days it was observed that this mass diminished in size. As there was no evidence of dissemination it was decided to explore the abdomen without further investigation, for it was quite clear that in any event he could not be left with an open cæcostomy, and if, as I feared, the growth could not be removed it was my intention to make a lateral anastomosis and to close the cæcum.

On March 9 the abdomen was opened by a vertical incision in the vicinity of the mass ; the abdomen was bulky, and, rather than expose the mass in its entirety, it was explored by palpation. It was large, rounded, and intensely hard, and I was in considerable doubt as to its nature. Still thinking that we had to deal with a growth, the liver was examined, but there were no secondary deposits. Incidentally the gall-bladder region was palpated and was found to be surrounded by what appeared to be a mass of adherent bowel and omentum. The incision was now enlarged to enable me to examine the mass more thoroughly and perchance to withdraw it from the abdomen for inspection. It was then apparent that there was something in the transverse colon just like a large bobbin, but it was so enormous that I thought more of an enterolith than of a gall-stone. Whatever its nature, the wall of the bowel was so tightly stretched over it that it was quite anæmic and the longitudinal bands were so spread out as to be merged and indistinguishable. The mass could not be moved, so an incision was made directly over it in the long axis of the gut. The moment the bowel was opened there was no longer any doubt as to its nature, for I saw that we had to deal with a gall-stone of unusual size. It required an incision about 2½ in. long to enable the calculus to be extracted without tearing the intestinal wall. On removal, it was noted to have a terminal facet, and this prompted a further search, when a second,

large enough, but much smaller, calculus was found lying in the bowel on its proximal side, and this was removed. There was very little fæcal escape, but what little muco-fæcal discharge occurred was exceedingly foul, as indeed were the calculi themselves.

Owing to the emptiness of the bowel the bed in which the gall-stone had lain could be carefully inspected. It was not ulcerated, but was mottled with curious pink areas which looked like a cutaneous surface and may have represented healed ulcers. The intervening mucous membrane was dark purple and with an appearance like the pile on velvet. The incised bowel wall was sodden and friable and in consequence a little difficult to suture. As the lumen was in no way narrowed, the incision was closed in the longitudinal direction, being fortified externally by neighbouring omentum. This operation was carried out under general anæsthesia and was followed by some shock, but the patient soon rallied, and in a few days passed some flatus per anum, though almost the whole of the intestinal contents continued to escape by the cæcostomy. About ten days later he was not so well, his appetite failed, and he developed symptoms of mental derangement amounting to delusional suspicion. On March 23 the cæcostomy was closed ; this interference again necessitated a general anæsthesia as the surrounding skin was so sore.

For a time the mental confusion improved, but by the end of two weeks it was so troublesome that as a therapeutic measure the patient was sent back to his home surroundings. There he soon improved, and by the end of April was able to be up and about, and has since been restored to his normal health. In the light of the disclosures of the operation, this patient was very closely questioned and was encouraged to search his memory, but he could not recollect ever having suffered from abdominal pain until the attack twelve months previously.

The calculi removed are certainly noble specimens. The larger stone when fresh weighed 5 oz. (4800 gr.) and measured 3 by $2\frac{1}{4}$ by 7 in. circumferentially ; even the smaller stone turned the scale at 1 oz. (960 gr.) and measured $1\frac{3}{4}$ by $1\frac{1}{2}$ by $4\frac{1}{2}$ in. circumferentially (*Fig.* 10). It is of interest to note that the calculi lost in weight as they dried and that their surfaces began to crack and peel. The illustration shows the exact size of the stones, but somehow they do not look either large enough or sufficiently massive. I suppose that only stereoscopically would they appear at their best, but even then, compared with their startling size when first removed, it would be as freshly caught salmon shimmering on the gravel by the river side to the same fish weltering on the marble slab of the shopkeeper ! Both calculi have found a fitting asylum in the Royal College of Surgeons Museum, but there they must make their neighbours feel at a disadvantage, for even in that galaxy of giants there are none that can approach them in magnitude. A search of the British literature, willingly made by Mr. S. Wood of the Royal College of Surgeons, did not unearth the record of any calculus approaching the larger stone in size, the nearest by weight being only 3 oz. and 5 drachms. Envious critics stoutly asserted that so large a concretion must be an enterolith, but Mr. T. W. P. Lawrence has kindly roughly assayed the stone for me, and finds it composed almost entirely of cholesterin and bilirubin pigments. He has no doubt but that it belongs to the genus Gall-stone.

In all these cases the question of the route by which such calculi enter the intestine always arises. I discussed this matter in a paper on " Intestinal Obstruction from Gall-stone " in 1927.[1] The point is so constantly raised

FIG. 10.—The calculi. The larger weighed 5 oz., and measured 7 in. in circumference. (Actual size.)

and is so interesting that I crave the permission of the editor and the indulgence of the reader for the repetition of the following paragraphs from that paper.

" The absence of previous symptoms of gall-stones is often a remarkable feature. When confronted with these cases people invariably say what a curious thing that the patient has never had a history of jaundice! As a matter of fact, gall-stones which are sufficiently large to produce intestinal obstruction do not often traverse the common duct, but find their way into the intestine by ulceration from the gall-bladder, either directly into the duodenum, or into the large bowel. Many years ago (1890) Courvoisier went carefully into this matter, and from post-mortem investigation of 36 cases he found that the common routes were as follows :—

Through the gall-bladder—
    Duodenal fistula twenty-five times.
    Ileum fistula once.
    Colon fistula once.
    Duodenal and colon fistula twice.
Through the common bile-duct seven times.

" In addition to the routes specified in this table I have seen the communication directly between the gall-bladder and the stomach, and cases are on record in which gall-stones have actually been vomited. In the College of Medicine Museum in Newcastle there is a specimen showing two gall-stones, composed mostly of pigment. The larger stone, which is dark green in colour, and measures $2\frac{1}{2}$ in. in length, was removed at operation from the stomach. The smaller one, which is brown and possesses a facet which fits the end of the larger stone, was found impacted at the lower end of the ileum, where it was the cause of intestinal obstruction. A fistula between the gall-bladder and the duodenum was demonstrated. These calculi were removed by my colleague, Mr. T. A. Hindmarsh, from a woman of 53 on whom he operated for obstructive symptoms. The patient made a satisfactory recovery, and was in good health a year later. There may be an opening into all three neighbouring viscera, and not uncommonly into both the small and the large bowel. Even when the stone traverses the common duct it probably does not usually find its way through the normal opening, but ulcerates from the ampulla directly into the bowel, and this is proved by an investigation of museum specimens. Among great rarities there are cases where the gall-bladder has burst into the urinary bladder or even the uterus. A knowledge of pathology, therefore, does not lead one to expect a history of jaundice in many of the cases, but it might be supposed that there would always be a history of a definite and perhaps severe gall-stone illness leading up to the passage of the stone into the bowel. As a matter of experience this is not by any means always so, and in my own series of eight cases such a history was entirely absent in two and indefinite in a third. Experiences of this sort made me pay particular attention to this matter, and I soon found that many patients with distended gall-bladder from obstruction of its neck or cystic duct have but few symptoms. In 1921 I operated on a female patient, 44 years of age, who gave a history that she had suffered from indigestion for two months, with an attack of discomfort and soreness over the upper abdomen four days before she came under my observation. The gall-bladder was distended and reached nearly to the umbilicus, yet the patient stated that 'she could hardly say there had ever been pain.' On opening the

abdomen 1 found an inflamed gall-bladder full of infected mucus, the result of the impaction of a large stone in its neck. More than once I have seen similar cases where the patient would not submit to operation. The swollen gall-bladder has subsided, sometimes suddenly, and all symptoms have ceased.

" In most of such cases the gall-bladder leaks into a hollow viscus, and the stone or stones may be gradually forced into the bowel by a slow process of ulceration, as shown in the illustration (*Fig.* 11). As soon as the tension in the gall-bladder is relieved, the symptoms disappear, and the process of the gradual extrusion of the stone into the bowel is unattended with symptoms. When the gall-bladder and neighbouring parts are examined in these cases, the viscus is found to be small, thick-walled and shrivelled, and the cystic duct commonly obliterated. The opening which first forms is not large enough to allow a stone of such size as would cause obstruction to enter the intestine, but after
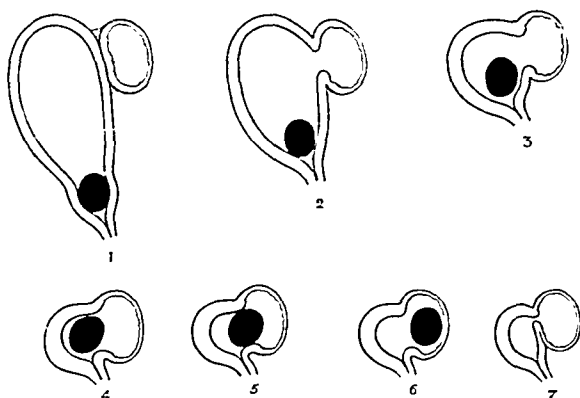
Fig. 11.—A series of diagrams showing how gall-stones may find their way into the hollow viscera. 1, A calculus is impacted in the neck of an inflamed and distended gall-bladder which has become adherent to the neighbouring bowel. 2, The gall-bladder has ulcerated into the intestine. 3, The gall-bladder drains freely into the bowel, and tension being relieved it commences to contract. 4, The process of contraction which is partly due to the development of scar tissue continues and the calculus is pressed against the opening in the bowel. 5 and 6, Show the transit of the calculus into the bowel. 7, The calculus has travelled on down the intestine, the fistula contracts, and the gall-bladder continues to shrivel.

a time the gall-bladder contracts on its contents and causes the stone to be pressed against the sinus, which is gradually enlarged by ulceration until it is big enough to let the calculus through. This is very well shown in a specimen in the College of Medicine Museum in Newcastle. [An illustration of this specimen was shown in the original paper.] The legend was as follows : ' The first part of the duodenum is laid open showing a large communication with the gall-bladder, which is occupied by a faceted calculus. Lying free in the bowel is the sister calculus, which became impacted in the ileum and caused the death of the patient from acute obstruction. The facets on the calculi exactly correspond.' In this specimen the two stones had evidently lain together with the broad facets in apposition, and the fistula is just of the exact size of the stone in the intestine. For some little time after the passage of the stone the fistula probably only slowly contracts, because in one or two

instances it has been found possible to pass the stone from the intestine back into the gall-bladder. In the specimens that I have had the opportunity of dissecting, although the stones were large (1⅞ by 1 in. and 2¼ by 1 in. respectively), the opening into the gall-bladder would only admit a forefinger and the margins were very rigid. The opening will still further contract after the stone has passed, though a communication, however small, probably always persists, forming the fistula bi-mucosa. Many experiences have confirmed this view of what happens, and all the stages of this process may be observed in the course of operations for gall-stone. *Figs.* 9 and 9A [in original paper] represent the contents of gall-bladders which I have removed. Any of these stones might have been the cause of intestinal obstruction had they found their way into the bowel. The calculus shown in *Fig.* 10 [in original paper] was removed from a suppurating gall-bladder in the case of a female patient 38 years of age. The inflamed viscus was very adherent to both the stomach and colon, and had there been no surgical intervention, it is highly probable that the large stone, which is just of the size to produce an intestinal obstruction, would have found its way by ulceration into the gastro-intestinal canal.

" Frequently when operating for gall-stones I have found communications between the viscus and some part of the bowel, and have elsewhere drawn attention to the way that these tend to be drawn out and become infundibuliform, then presenting considerable danger, for they may be inadvertently treated as simple adhesions. In July, 1909, I operated on a female patient, age 32, for the removal of a large ovarian cyst. A mass felt in the gall-bladder region was investigated, and a large stone found lying between the remains of the gall-bladder, the hepatic flexure of the colon, and the stomach, both of which had been opened by this process of slow ulceration. The calculus was removed and the patient made a good recovery. I made every endeavour to obtain a history of an attack of abdominal pain or previous abdominal illness, but she assured me that there had been none.

" This tendency of foreign bodies to find their way into the hollow viscera seems very general in pathology, and is certainly not limited to gall-stones. Some years ago I published (*The Lancet*, May 6th, 1916) some cases of ' Foreign Bodies in the Bladder resulting from Gunshot Wounds ' and gave examples of foreign bodies which had slowly and gradually ulcerated into that viscus. Recently I have had a further remarkable example in the person of an ex-soldier who was wounded in May, 1917, portions of a shell being embedded in the left buttock and thigh. Two operations were performed for removal of fragments, and later for secondary hæmorrhage. He eventually made a good recovery and was discharged from the Army in July, 1918. He soon regained his usual good health, and was able to resume employment in a bank. There was never the slightest suggestion of bladder trouble until April, 1925, when he began to have frequent painful micturition. These symptoms continued until July, when he was admitted under my care. Cystoscopic examination disclosed the piece of shell, which was removed (it measured 1½ by ¾ in.). There was only a slight cystitis, and no scar to indicate where the fragment had entered the bladder. There can be little doubt that it lodged in the soft parts outside the bladder at the time of the casualty, and

that by a slow process of ulceration it was extruded into the viscus, probably about April, 1925. Between then and the time of its removal the track by which it entered the bladder had closed, for in the process employed by nature ulceration and repair go hand in hand."

In the present case the site of impaction is unusual, for as a rule calculi causing obstruction are arrested in the small bowel. I have now operated on ten occasions for gall-stone ileus, and only in two was the impaction in the large bowel. Calculi of sufficient size to block the large bowel must be very unusual, and any stone that has passed the ileocæcal valve is probably assured of an unimpeded passage through the large bowel and to the world beyond. But I know of a case in which such a calculus stuck at the anus and had to be manually extracted. Even stones which reach the lumen of the large bowel direct must be of large size to be arrested, though, as in the small intestine, spasm may play a part, and I believe this is especially likely to occur in the sigmoid. This case adds another recovery to my admittedly fortunate series, making 8 recoveries out of 10 operations for this very serious condition.

REFERENCE.

[1] GREY TURNER, G., *Post-Graduate Med. Jour.*, 1927, Feb.–March.

THE EARLIEST SURGICAL TREATISE.*

By WARREN R. DAWSON, F.R.S.E.

ANCIENT Egypt is the starting-point in all studies in the history of medicine. From the literature of that venerable civilization that flourished on the banks of the Nile the earliest known medical documents have come. All that has hitherto been written of the history of medicine in ancient Egypt is mainly based upon a single document—the well-known Ebers Papyrus. This venerable manuscript was found in a tomb in 1862 together with another medical papyrus (with which this paper is concerned), a book of mathematical tables and problems (now well known as the Rhind Mathematic Papyrus), and some other documents. The two medical papyri passed into the possession of a certain Edwin Smith, an American resident in Luxor who dealt in antiquities but at the same time took an intelligent interest in them and made considerable attempts to learn the language in which the texts and inscriptions were written. The longer of his two medical papyri he sold, a few years after its acquisition, to a German Egyptologist, Georg Ebers. The delighted purchaser named his newly-gotten prize after himself, although what has become generally known as the Ebers Papyrus is always referred to in Egyptological literature prior to his purchase as 'Papyrus Smith'. Ebers published a facsimile of his medical manuscript with an introduction and glossary, but he wisely did not attempt a translation. I have elsewhere given an account of this and of the other known Egyptian medical manuscripts† : it is therefore needless to say anything further of them now beyond quoting a single paragraph from my former account :—

The Ebers Papyrus is not a book in the proper sense of the word ; it is a miscellaneous collection of extracts and jottings collected from at least forty different sources. To call it, as Ebers did in his publication, a Hermetic Book, is entirely to mistake its nature and purpose. It consists mainly of a large collection of prescriptions for a number of named ailments, specifying the names of the drugs, the quantities of each and the method of administration. A few sections deal with diagnosis and symptoms, another passage is physiological in character and describes the action of the heart and its vessels, and the concluding portion is surgical, being concerned with the treatment of wounds and suppurating sores. Freely interspersed amongst these elements are spells and incantations.

Egyptologists familiar with the intricacies of the language have long recognized that the corruption of the text, together with its intrinsic linguistic difficulties, is such as to make an intelligible English translation of the Ebers Papyrus wellnigh impossible. This justified caution, however, has not

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deterred others. knowing little or nothing of the hieratic script and of the language imparted by it. from rushing in where Egyptological angels feared to tread. and the result has been the production of several so-called 'translations' that are composed mainly of guesswork. and in the violence they do to grammar and syntax are mere travesties of the originals they profess to interpret. Unhappily it is on these data (and on one 'translation' in particular. which unfortunately has recently been given a new lease of life) that the medical historians have mainly drawn. with the result that an erroneous notion of the real nature of Egyptian medicine has been imparted to former generations of students. which the present generation is making some attempt to correct. The Ebers Papyrus and its kindred documents—the medicomagical papyri of London. Berlin. Leiden. California. and elsewhere—may now be dismissed from our attention. as they are merely miscellaneous collections of recipes and incantations, and (save for a few passages) they have no right to be considered as scientific medical books.

The other of the two medical papyri which belonged to Edwin Smith remained in his possession all his life. but it was not published.* After his death in 1906 the manuscript passed into the hands of the New York Historical Society. A few years ago this body invited Professor James Henry Breasted to study and edit their papyrus, and a preliminary examination so impressed Professor Breasted with the interest and importance of the text that he undertook to publish it *in extenso*. He has recently published the result of his labours in two large volumes : one containing a photographic reproduction of the hieratic text and a transcript into hieroglyphs, the other an introduction, translation, and commentary.†

It is most fortunate, not only for Egyptology but for the history of medicine, science, and human thought in general, that this important document has fallen into the hands of so competent a translator. Professor Breasted's translation may be relied on, in the main. as thoroughly accurate and sound. although naturally there are some points upon which opinions may differ. In addition to the literal translation with its elaborate philological commentary, Professor Breasted has provided for medical historians and others unacquainted with the ancient Egyptian language, a running translation of the whole text, thereby imparting to readers a general idea of the purport of the document without involving them in philological and other technical discussions.

The Edwin Smith Papyrus contains two entirely distinct groups of writings. The first is a surgical handbook. and the second is a batch of miscellaneous recipes and charms similar in nature and content to those

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* In 1864 Edwin Smith had some intention of publishing the papyrus, but the project never materialized. (Brit. Mus. Add. MSS., 31,285, fo. 130.)

† *The Edwin Smith Surgical Papyrus.* Vol. I, Hieroglyphic Transliteration, Translation and Commentary. pp. xxiv + 596, 8 plates, 4to. Vol. II, Facsimile Plates (22 collotype, and 22 line plates), folio. University of Chicago Press, 1930.

Professor Breasted had previously published three preliminary accounts of the papyrus: (1) *New York Historical Society, Quarterly Bulletins*, 1922. April, vi, pp. 4–31. (2) *Recueil d'Etudes égyptologiques dediées à la Mémoire de Jean-François Champollion*, 1922, pp. 385–429. Paris. (3) *Bulletin of the Society of Medical History of Chicago*, 1923, Jan., iii, pp. 58–78.

of the Ebers and other papyri. The latter are written on the back of the roll, the face of which is occupied by the surgical book. The main (surgical) text differs wholly from those of the previously known medical papyri (except a passage in Ebers which is similar in nature and arrangement), in that it is a continuous one and as such it is definitely entitled to be called a book; it is indeed by far the earliest known surgical treatise, and it deals systematically with the examination and treatment of wounds and fractures. Unfortunately the text is incomplete, and it breaks off abruptly in the middle of a sentence. The copyist apparently tired of his work, or perhaps he had borrowed the original and was obliged to return it to its owner before he had had time to complete his transcript. Having a large extent of blank papyrus left on his hands, he filled the free space with matter of a very different kind.* As it stands, the text comprises the details of forty-eight cases of wounds and fractures, classified in the 'head-to-foot' sequence that is common in Greek and later medical books. The injuries described begin with the top of the head and, proceeding in orderly succession downwards, reach as far as the thorax. At this point the text breaks off, but it is fair to presume that the original book, of which the Edwin Smith Papyrus is but a partial copy, completed the exploration of the body down to the feet.

The cases are presented in a systematic fashion, and each contains the following elements: (1) Title; (2) Examination; (3) Diagnosis; (4) Verdict (one of three alternatives); and (5) Treatment. A valuable feature of this text is the fact that in many cases glosses or explanatory notes have been interpolated, and these provide us with much valuable information and make intelligible to us expressions that would otherwise have remained obscure and meaningless idioms. The cases are all concerned with wounds, fractures, dislocations, and similar injuries, and the treatment is, generally speaking, appropriate and rational throughout, and contrasts markedly with the strange and often grotesque remedies familiar in the Ebers and other papyri. It must be remembered, however, that the Edwin Smith Papyrus deals with injuries of human, and therefore of intelligible, origin, and not with sickness and disease, of which the causes were entirely unknown to the ancients, and, being regarded by them as of supernatural origin, were treated by magical rather than by rational means.

The general coherence and intelligibility of the text of the Edwin Smith Papyrus has made its translation a far easier problem than is the case with regard to the other medical papyri. But the text is by no means free from obscurities and difficulties, and it abounds, moreover, in corruptions. To make its general arrangement clear, I cannot do better than select two specimens out of the forty-eight cases detailed in the document, and in making the choice I have selected two which are short in extent and relatively free from difficulties. Most of the cases are much more detailed, but are too long for convenient quotation. Case 21 is a good one for our

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* Papyrus was expensive material and was never wasted. It is consequently usual to find the space not occupied by the main text subsequently employed for other purposes. In this way it is common to find in a papyrus mainly devoted to a religious or literary text, scraps of accounts, journals, and other memoranda covering any free space that may remain.

purpose, and I here transcribe Professor Breasted's rendering of it (op. cit., p. 287) :—

Instructions concerning a split in his* temple. If thou examinest a man having a split in his temple, shouldst thou find a swelling protruding on the outside of that split, while he discharges blood from his nostril and from his one ear having that split, (and) it is painful when he hears speech, because of it :
Thou shouldst say concerning him : " One having a split in his temple, while he discharges blood from his nostril and his ear having that injury. An ailment with which I will contend."
Thou shouldst put him at his mooring stakes, until thou knowest he has reached a decisive point.

The five elements already referred to will easily be recognized. The first is the title, which always begins " Instructions for ", etc. ; the second is the examination, beginning " If thou examinest " ; the third is the diagnosis, beginning " thou shouldst say concerning him " ; the fourth is the verdict, " an ailment with which I will contend " ; and the fifth and last is the treatment. In this case the treatment consists of the remarkable expression " thou shouldst put him at his mooring stakes ". This is an idiom which occurs in several of the cases, and it would be wholly unintelligible to us were it not for the explanatory gloss which is given in Case 3, where the phrase first occurs. The gloss explaining the phrase " put (or moor) him at his mooring-stakes" is thus translated by Professor Breasted (p. 139) : " As for 'moor (him) at his mooring-stakes', it means putting him on his customary diet without administering to him a prescription." I cannot, however, help thinking that Professor Breasted has misunderstood the significance of the phrase. Surely to moor a man to a stake is a metaphorical way of expressing the idea of confining or restricting him, and although the word (*metr*) which Professor Breasted translates 'customary' may have that significance in certain contexts, its usual meaning rather implies the notion of limitation by rule, restricting. *To put a patient on his customary diet without prescribing any medicine is surely tantamount to giving no treatment at all.* The implication, therefore, of the idiom is that in cases that are to be treated by thus 'mooring' the patient, mere external applications† are of no use, but the patient's health and strength must be restored not by his free and customary diet, but by a *special* diet. In such cases, external applications must be used until sufficient improvement by the first method has been accomplished. That, at least, is how I understand the text.

After this excursus, another specimen case may be quoted. Case 22 deals with a compound comminuted fracture of the temporal bone, and it reads as follows (Breasted's translation, p. 290) :—

Instructions concerning a smash in his temple. If thou examinest a man having a smash in his temple, thou shouldst place thy thumb upon his chin (and) thy finger

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* It may here be noted that in all Egyptian medical texts, the organs are never spoken of in the abstract as *the* heart, *the* stomach, etc., but as *his* heart, *his* stomach, etc. There is always a hypothetical patient whose case is being discussed.

† In the Edwin Smith Papyrus ' prescriptions ' are always external applications, not doses.

upon the end of his ramus, so that the blood will flow from his two nostrils (and) from the interior of his ear having that smash. Cleanse it for him with a swab of linen until thou seest its fragments (of bone) in the interior of his ear. If thou callest to him (and) he is speechless (and) cannot speak,

Thou shouldst say concerning him : " One having a smash in his temple ; he discharges blood from his two nostrils and from his ear ; he is speechless ; (and) he suffers with stiffness in his neck. An ailment not to be treated."

As for : " The end of his ramus ", it means the end of his mandible. The ramus ('m'.t), the end of it is in his temple just as the claw of an 'am'e-bird grasps an object.

As for : " Thou seest its fragments (ws.t) in the interior of his ear ", it means that some of the fragments (ws.t) of the bone came away to adhere to the swab which was introduced to cleanse the interior of his ear.

In this case the third form of verdict is used, "an ailment not to be treated"—that is to say, the physician recognizes that the injury is of so serious a nature as to be beyond his powers, and that recovery cannot be expected. Of the second and third verdicts, the two cases quoted provide examples. The first form, "an ailment which I will treat" is used in simple cases where the practitioner is confident of a cure ; the second implies a case that must be seriously tackled and may be successful ; while the third is a frank admission that the surgeon considers the case to be hopeless. The first verdict occurs twenty-nine times, the second eight times, and the third fifteen times in the papyrus.*

The case just quoted has two explanatory glosses† : the first explains the meaning of the phrase " the end of his ramus ", the forked head of which, terminating in the condyle and the coronoid process is likened to the claws of a bird. The second gloss, by its reference to splinters of bone, makes it clear that a case of comminuted fracture is envisaged.

These two cases are, as I have already said, selected solely for their brevity. Some are very long and detailed, and in the elaboration of symptoms and diagnosis they reveal a spirit of real scientific observation and inquiry. Such, for instance, are the observations on the brain. As an organ, the Egyptians seem to have attached little or no importance to it : the heart was believed by them to discharge the functions that later ages recognized as belonging to the brain. In the Edwin Smith Papyrus, however, there are recorded some notes on the brain itself. It is there recognized that the cerebral hemispheres are enclosed in a membrane. This fact could not have been learned from the process of removing the brain during the manipulations of mummification, because the custom of taking away the brain was not introduced into the technique of embalming until the Eighteenth Dynasty, long after the composition of the Edwin Smith Papyrus or its prototype. In mummies of earlier periods the brain was invariably left *in situ* and no attempt was made to remove it. Nor could the embalmers, who in the course

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* The total number of verdicts is thus fifty-three in forty-eight cases. This is accounted for by the fact that two verdicts are given (in alternative circumstances) in Cases 7, 8, 34, 37, and 47, and none in Case 9. In Case 34, the second verdict should obviously be Verdict 3, and not Verdict 1 as erroneously stated in the manuscript.

† Actually there are three glosses in this case, but for the sake of brevity I have omitted the third.

of their craft dragged out the brain piecemeal through a forced passage hewn in the ethmoid or sphenoid processes, be aware of its appearance as a whole. The remark, therefore, in the Edwin Smith Papyrus, that the brain is characterized by a series of convolutions " like those corrugations that form on molten copper ", must have been based upon the actual observation of the organ exposed by an extensive gash in the skull. A reference is also made to a wound that lacerates the " membrane enveloping his brain " so that it " breaks open his fluid in the interior of his head "—an allusion to the meningeal membranes and the cerebrospinal fluid.

Furthermore, many interesting observations are made as to the effects of brain injury : the loss of control over various parts of the body, the tension of the facial muscles, giddiness, and other manifestations. Professor Breasted, however, seems to me to be making too generous a claim for the medical knowledge of the Egyptians when he understands that their physicians recognized these symptoms as proceeding from injury to the *brain*. It seems to me that they are implicitly ascribed to injuries to the *skull*, without the definite knowledge that the brain itself is the ultimate cause of the observed effects. In some cases, indeed, the ancient surgeon seems to have been at fault : there is evidence, for instance, that it was observed in the case of an injury to the skull involving the brain, that the eye is affected on the same side as the injury to the skull, and partial paralysis of the leg resulting from a head injury is also noted as being on the same side as the wound in the skull that caused it. There are two possibilities open here : either that the text is corrupt, and instead of reading 'on the same side', the words 'on the opposite side' were originally written ; or, alternatively, the observer may have had before him a case of *contre-coup*, from which he wrongly generalized. Be this as it may, it is most interesting to find, in a book written more than thirty-five centuries ago, even the bare observation (be it rightly or wrongly stated), that injury to the skull (brain) produces manifestations in areas far removed from the seat of the wound, and that the side of the body in which such manifestations occur should be noted in relation to the side of the head that is injured.

Another observation of great interest is that recorded in Case 31, where it is noted that injury to the spinal column (in this case a cervical dislocation) may cause a prolonged priapism, sometimes followed by *emissio seminis*. In the description of the symptoms of the same case, it is also said that " his flesh has received wind ". This is evidently a reference to meteorism, when, owing to intestinal paresis, undischarged gases cause inflation of the abdomen.

Throughout the papyrus, the details of such cases are sufficiently fully stated, or explained in the glosses, to enable us to recognize them and to name them in terms of modern anatomy, physiology, or pathology with considerable precision.

Some of the cases are of simple flesh wounds that do not involve the bones or indeed any tissues other than the superficial. Amongst such cases are wounds in the scalp, chin, brow, nose, external ear, and lips. Injuries that are more deeply seated include a wound in the throat that penetrates the gullet, a gaping wound in the shoulder associated with inflammation and

fever, and gaping wounds in the head and neck penetrating the bones of the skull and cervical vertebræ respectively. The bone injuries mentioned include : *perforation* of the skull and its sutures, of the maxillary and zygomatic processes, and of the temporal bones; *fractures* (simple, compound, or comminuted) of the skull, of the nasal cartilage, of the maxillary, zygomatic and mandibular bones, of the humerus, of the clavicles, of the ribs, and of the cervical vertebræ ; and *dislocations* (and sprains) of the mandible, of the cervical vertebræ, of the clavicles, and of the sternocostal articulations. These, together with localized tumours and abscesses, make up the forty-eight cases of the Edwin Smith Papyrus.*

A few words may now be said as to the methods of treatment employed. The retracted edges of gaping wounds, if small, were drawn and held together by means of adhesive plaster, or, if large, by stitching. Minor wounds were treated by simple bandaging. Raw meat was often used (as it is to-day) as an external application to reduce swelling or inflammation, but it is specifically stated that it is to be used only on the first day. An ointment, the basis of which was honey, was freely used, often applied on swabs. For bone injuries, reduction of dislocated or fractured bones was frequently resorted to, with or without the subsequent use of splints. The rigid splints (of which actual specimens have been found) were tubular cases of palmfibre,† but in some cases supports, in the form of stiff linen rolls, were used. The actual surgical treatment was often accompanied by special diet, or the treatment in some cases was by diet in preference to therapy, a point that has already been alluded to in this paper. The ointments generally consisted of honey mixed with pounded herbs or with decoctions of herbs. The latter are often quite valueless by modern standards, although amongst them may be recognized a few that have well-marked astringent or antiseptic properties. The choice of the herbs and other drugs was still largely influenced by magic.

Reference has already been made to the glosses in the Edwin Smith Papyrus. They are of great interest and were evidently intended to explain the significance of expressions or terms that were considered to be too technical to be understood without such aid or that had become obsolete since the book was first composed. Twenty-nine out of the forty-eight cases have one or more of these explanatory glosses, of which there are altogether sixty-nine. It may be noted in passing that certain of the passages in the Ebers Papyrus that contain anatomical or physiological particulars are accompanied by glosses; but that long document (physically nearly seven times as long as the Edwin Smith Papyrus) contains altogether only twenty-six glosses, many of which have been displaced by ignorant copyists and thereby rendered scarcely intelligible. The glosses in the Edwin Smith Papyrus show that their composer was actuated by a really scientific interest in the cases before him. He often goes into details regarding the hopeless cases that he could not remedy, which shows that his interest did not cease merely with the

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* There are twelve cases of flesh wounds, and thirty-three of injuries to bones and articulations.
  † G. ELLIOT SMITH " The Most Ancient Splints ", *Brit. Med. Jour.*, 1908, March 28, p. 732.

practical side of treatment, but that his curiosity was stimulated to investigate what he could not cure. This fact indicates an approach towards the Hippocratic method. In his editorial introduction to the Hippocratic *Epidemics*, Mr. W. H. S. Jones says* :—

> Of the forty-two cases, twenty-five end in death, very nearly 60 per cent. The writer's aim is not to show how to cure—treatment is very rarely mentioned—but to discover the sequences of symptoms, to set down the successes and failures of Nature in her efforts to expel the disease. The physician is acting, not *qua* physician, but *qua* scientist ; he has laid aside the part of the healer to be for a time a spectator looking down on the arena.

Of the text itself of the Edwin Smith Papyrus, the aim is purely practical : its function is to describe symptoms and to indicate treatment. To the original author of the book, who probably lived at the time the Pyramids were being built, the lofty motives of the Hippocratic School cannot be attributed, but the glosses show that upon some later commentator was dawning the inspiration to make original research into causes and effects. In his notes, garbled and corrupt as they often are in the form in which they have come down to us in this the earliest surviving copy of them, are to be discerned the germs of that spirit of philosophical inquiry that only the genius of the Greeks with their powers of abstract thought (a power totally lacking in the Egyptians, gifted though they were) could bring to the stage they actually attained—that of forming the foundation upon which many generations of thinkers and inquirers have erected the edifice of modern medicine.

The Edwin Smith Papyrus, containing as it does the earliest practical handbook of surgery together with the notes of an ancient forerunner of Hunter, can justly claim to be the oldest scientific book. Professor Breasted declares that it destroys the general belief, in which he formerly shared, that Egyptian medicine had its origin in magic. With this general opinion I find it impossible to agree. It does not in the least detract from the importance and interest of the Edwin Smith Papyrus to recognize that while it undoubtedly affords evidence that a serious attempt was being made to understand the elements of anatomy and physiology, yet it must be clearly borne in mind that it is a book that deals only with wounds and fractures—injuries of palpable and intelligible origin—and not with diseases, for which perforce magical methods had to be employed, because the causes of illness and disease were to the ancients invisible, impalpable, and unknown. A wound or an injury caused by a fall or other accident, or by a weapon or a tool, was well understood and treated by rational means ; but the causes of headache and fever, of skin diseases or swellings, and of countless other maladies, were wholly mysterious and attributed to supernatural agencies. Two brothers might on the same day come before the doctor at Memphis or Thebes, the one for treatment of a dagger-wound in his breast, the other for a painful and irritating rash affecting the same region of the body. The cause of the one was self-evident ; the cause of the other was a mystery, and the treatment in the two cases differed in its nature accordingly.

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* *Hippocrates*, i, p. 144 (Loeb Classical Library, 1922).

Magic never lost its hold upon medicine, and it has retained its grip even to this day to a far greater extent than is generally recognized. Whilst the beginnings of real research were lifting medicine out of the domain of the magician, the treatment of common ailments still and for long ages continued to come under his eye. The medical and the magical marched side by side, and the same age produced both the Edwin Smith and the Ebers papyri, with their widely-differing contents. Indeed, on the back of the Edwin Smith Papyrus itself there is written a collection of charms and prescriptions similar in character to those that fill page after page in the other so-called 'medical' papyri. The ancient owner of the Edwin Smith Papyrus saw no incongruity in having in one and the same note-book elements that appear to us of to-day as absolutely antagonistic in nature and content. One might imagine, as a parallel, a modern student taking simultaneously and equally seriously the utterances of Paracelsus and of Hunter. I will conclude by a brief reference to these miscellaneous jottings that fill the verso of the Edwin Smith Papyrus.

There is first a series of eight incantations against plague. These are grouped under such headings as " Incantation for exorcising the wind of the pest of the year ", or, " Another for exorcising the plague-bearing wind,* the demons of disease, the malignant spirits, messengers of Sekhmet.† " These are all charms or incantations to be recited over images of gods or other talismans, and are purely magical. The next item is a prescription for a female complaint, and as I cannot agree with Professor Breasted's translation of it, I will offer a rendering of my own :—

If you examine a woman who suffers in her stomach, so that for her the menses do not come, (and) if you find something in the upper part of the vagina, you say concerning her : " It is a clot of blood‡ in the womb." You prepare for her balsam, $\frac{1}{16}$; grease, $\frac{1}{8}$; sweet ale, $\frac{1}{8}$; warmed and drunk for four days ; besides preparing for her (the prescription for) the discharge of blood, (namely) oil, cumin, stibium, sweet frankincense. Mix and anoint the pubes§ therewith frequently.

As I understand the passage, it is clearly the treatment of irregularity in menstruation due to clotting. The symptoms are nausea and local pain. For the relief of the former an emetic is prescribed, and for the latter an ointment to be rubbed on the lower part of the abdomen to relieve the pain in the vagina and to provoke the flow by massage.

After this, another prescription for menstruation follows, this time employing a fumigation. The concluding recipes are all pure quackery and differ little either in substance or in kind from the boldly-advertised trash of the seventeenth and eighteenth centuries in Europe, which under a thinly disguised exterior is still prevalent to-day. The titles of these recipes alone sufficiently indicate their character : " Recipe for Transforming the Skin ",

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* That plague was carried by the air remained one of the main tenets of medical opinion for many centuries later. It was the dominating principle that ordered the closing of windows and the lighting of fires in the streets in the Great Plague of 1665.

† A malevolent goddess.

‡ *Shen'w n snf*, lit., ' an obstruction of blood '.

§ Reading *Kns* for *Ks* as written in the MS. in error.

" Recipe for Beautifying the Face ", and lastly, " A book for Transforming an Old Man into a Youth ". The quest of personal loveliness and eternal youth is as old as the cradle of civilization.

We must not, however, allow the quackery of the verso of the Edwin Smith Papyrus to prejudice our minds or to detract from the rational and valuable surgical treatise of the recto. A careful study of the forty-eight cases that have survived must convince an unprejudiced reader that the Surgical Book of the Edwin Smith Papyrus has a just title to be known and respected as the earliest scientific book that time has bequeathed to posterity. Medical historians must revise their estimate of Egyptian medicine in the light of this new accession.

A CASE OF TRANSPLANTATION OF THE RIGHT URETER INTO THE CÆCUM AND OF THE LEFT INTO THE SIGMOID FIVE YEARS AFTERWARDS.

By C. E. L. BURMAN, Pietermaritzburg, Natal.

As far as I am aware the cæcum has not been used as a reservoir for the urine, and it would appear that there are certain advantages to be gained by such a procedure in preference to the recognized sigmoid route : (1) The chances of ascending infection, although considered not tenable to-day, are certainly less than in the sigmoid. (2) The urine passes into a more or less definite receptacle. (3) Absorption of water takes place during transit of urine along the whole length of the large bowel, with consequent less desire to pass urine. (4) There is no chance of solid contents interfering with the flow of urine from the kidney, as the contents in this area are fluid. (5) The cæcum is easily fixed in a convenient position. (6) The appendix stump orifice is a convenient and comparatively easy and safe method of anastomosis by means of the ink-well method. (7) There appears to be no irritation when the urine passes into the cæcum, as the acidity is quickly neutralized by the alkaline contents from the ileum.

The following case supporting the above statements seems to be justified :—

HISTORY.—In January, 1926, J. V., female, age 16 years, had pains in the abdomen for two days and was then taken by her people to see a European doctor, who drew off a large quantity of urine and told her she was pregnant and in labour. The patient herself was not aware that she was pregnant until told by the doctor. This may seem a difficult statement to believe, but in the raw native I have seen it more than once. The labour was apparently difficult, and craniotomy was carried out. She was on her back for two months following the confinement. I saw her on June 25, 1926, when she complained of being constantly wet and of inability to hold her urine.

ON EXAMINATION.—Considerable ulceration of the posterior and lateral walls of the vagina was revealed and a large hole in the anterior wall of the bladder. By retracting the sides of the hole the ureteric orifices could be plainly seen. The whole urethra except for the external urinary meatus was entirely absent. A plastic operation was carried out and a catheter tied into the bladder. The catheter came out and was not replaced, with the result that the newly constructed urethra gave way and the patient was no better off. Examination on July 16 showed that even the small piece of urethra had been obliterated and the external urinary meatus was represented by a dimple. It was decided that the only possible solution was a transplantation of the ureters, and the following operation was carried out on the right ureter the same day.

FIRST OPERATION (July 16, 1926).—An oblique muscle-cutting incision was made in the right iliac region of sufficient length to give good exposure of the cæcal and ureter area. The ureter was isolated and traced down to the entrance at the bladder and excised at the ureteric orifice and clamped at the extreme end. The hole in the bladder was closed and the ureter brought out of the wound and covered with gauze. An incision was then made in the posterior parietal peritoneum extending from the ileocæcal junction along the brim of the pelvis as far as the caput cæci. The appendix was clamped and removed in the usual way and the stump opened up. The ureter was brought through the tunnel and inserted, after freshening up the end, through the orifice of the appendix stump for about 2 in. into the cæcum. The peritoneum was invaginated all round the ureter and secured by four interrupted sutures of Pachenstecker thread catching up the peritoneum and ureter with each suture. Another row of interrupted sutures was inserted around this area so that the ureter was ink-welled into the appendix stump. The cæcum was fixed by suturing it into the incision made into the posterior parietal peritoneum. The incision for isolation of the ureter was closed with a few sutures, a drain being placed in the operation area, and the abdomen closed.

AFTER-PROGRESS.—The drain was removed on the third day after operation. Recovery was uneventful, and the patient was discharged from hospital on Aug. 2. Nothing was heard of her until Oct. 4, 1929, when she reported for examination. Her general condition was excellent, but she still complained of being wet—the result of the left ureter. Operation was advised and accepted.

SECOND OPERATION (Oct. 8, 1929).—Indigo-carmine (2 c.c.) was injected intravenously just before the administration of the anæsthetic. The cæcum was exposed through an independent incision on the right side and punctured with a needle with a view to ascertaining whether coloured urine was present in the cæcum, but none was obtained. The left ureter excreted coloured urine. An oblique muscle-cutting incision was now made in the left iliac region and the abdomen opened. The right kidney was first palpated and found normal in size and consistency. Transplantation of the left ureter into the sigmoid was now decided on, but the ureter could not be located and the operation was abandoned. The patient went home *in statu quo* on Oct. 22.

AFTER-PROGRESS.—The girl's whereabouts could not be traced until December, 1931, when I was able to have her admitted to Grey's Hospital, Pietermaritzburg. On Dec. 5 examination of the vagina was undertaken without an anæsthetic, but all that could be made out, owing to the tenderness and pain produced, was a sodden condition of the vulva and inner sides of the thighs, a marked contraction of the vaginal orifice and vagina, ulceration of the lateral and posterior walls, and that the urine was welling out of the bladder and forming a puddle in the vagina. The urinary meatus was represented by a small depression drawn over to the left side. On Dec. 8 radiograms of both kidneys were taken and the radiologist reported : " Gas shadowing obscures to some extent the right side (upper half). Dye appears in both pelves. The appearances suggest the presence of a double ureter on the right side (upper half). Slight diffuse opacity in the cæcal and ascending colon suggesting the free flow of uroselectan into the cæcum."

THIRD OPERATION (Dec. 10, 1931).—The old scar on the left side was excised and the abdomen re-opened. Considerable omental adhesions and matting of the sigmoid were encountered. The right kidney felt normal. The

FIG. 12.—Two minutes.
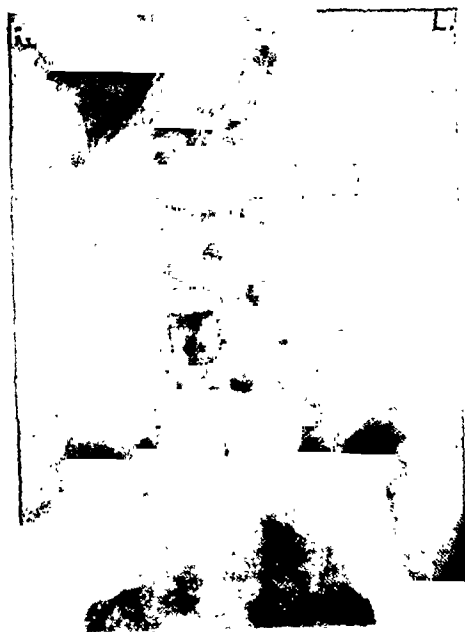

FIG. 13.—Five minutes.
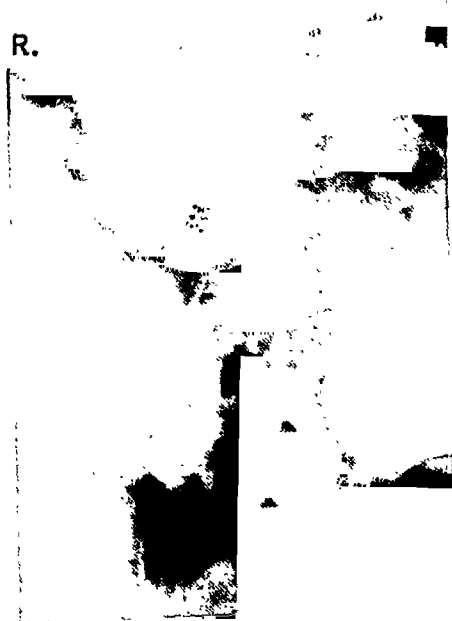

FIG. 14.—Fifteen minutes.

FIG. 15.—Forty-five minutes.

ureter was again found difficult to identify, but it was finally located some-what farther out than usual and traced upwards and downwards until a good 3 in. was isolated. The sigmoid was mobilized and the ureter transplanted by using a combination of Coffey's and Stiles's methods. No ureteral catheter was used, but a bed in the sigmoid was prepared by an incision of the peritoneal and muscular coats for about 2 in. and the ureter drawn through a small incision at the distal end and fixed into the bowel. Two further sutures were passed through the outer wall of the ureter and the peritoneum of the sigmoid, and the bed and ureter were finally buried with a continuous suture. A drain was passed down to the area and the abdomen closed.

AFTER-PROGRESS.—Uneventful until the sixth day following operation, when the patient was suddenly taken ill with an acute pain in the left axillary region with a rise of temperature to 102·6° and a pulse-rate of 120. The next day she complained of pain over the right side of the abdomen and distension. A slight watery dis-charge was noticed on the dressing, but a fair quantity of urine was passed by the rectum. A large poul-tice was applied for one hour. Relief was almost immediately obtained by the passage of flatus, faeces, and urine and diminution of abdominal disten-sion. The watery discharge, now more suspicious of urine, was more profuse, but it quickly grew less, and by Dec. 27 the patient was dry. The daily output of urine, from now onwards, was 50 oz. Tolerance varied from one to five hours. The wound was soundly healed.

Jan. 3, 1932.—Examination per vaginam was carried out by the in-sertion of a gauze soaked in 20 per cent cocaine solution, and it was found

FIG. 16.—Seventy-five minutes.

that the ulceration was considerably improved. The vagina was still markedly contracted, and the hole in the anterior wall of the bladder admitted quite easily the forefinger, allowing digital examination of the bladder. The failure of the original plastic operation was undoubtedly due to the inability to recon-struct the urethra.

Jan. 5.—Intravenous pyelography was carried out, and the report from the radiologist stated : " Slight excretion of the dye is noted in the left kidney 2 minutes after injection. At the 5-minutes examination slight tendency to hydronephrosis is seen in the left kidney and marked hydronephrosis in that of the right. The right ureter is not seen filled with dye in 15 minutes, but the pelves and calices are well defined. At the 45-minutes examination optimum concentration of the dye is present in the left side and marked kinking

of the ureter (dilated) is evident on the right side. The course of the ureters is seen in their entirety on both sides." (*Figs.* 12–16.)

Jan. 16, 1932.—The patient was discharged from hospital in excellent health (*Fig.* 17) and went to her own home up country. At the end of the

Fig. 17.—*Condition of patient on discharge from hospital.*

month she was suddenly taken with purging and vomiting, and from this she died on Feb. 3. There was no post-mortem.

COMMENT.

The marked dilatation of the right ureter with kinking and the associated hydronephrosis noted after pyelography following operation as compared with the pyelogram before operation suggests that the acute attack of pain complained of in the right side on the sixth day after the last operation was the cause of this unfortunate accident. The reason of this appears hard to explain as the patient had been perfectly well ever since the transplantation in 1926. It may be that the somewhat severe reaction which followed the administration of liquorice powder on the sixth day to obtain bowel evacuation caused an upheaval in both the recent and previous operation areas.

LEUCOPLAKIA IN THE URINARY TRACT, WITH A REPORT OF A CASE.

By H. P. WINSBURY-WHITE,

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LEUCOPLAKIA involving the urinary tract is uncommon, but of recent years many more cases have appeared in the literature than were formerly reported, suggesting that the disease at one time often escaped notice. Writing in 1929 Patch[1] found 123 reported cases involving the kidneys. ureters, and bladder.

CASE REPORT.

The following are the facts relating to a case which came within my personal experience :—

HISTORY.—The patient, a married man, age 41 years, with three children, was first seen by me in February, 1930, complaining of increased frequency of micturition, and discomfort at the end of passing water. In 1913 he had hæmaturia for three months. He was admitted to St. Thomas's Hospital. A note from the Surgical Registrar about his condition at this time is as follows : " In 1913 this patient had excision of a so-called papilloma of the bladder, the microscopical report of which was—chronic inflammatory. He was in again in 1921, when a cystoscopy revealed a bright red mucosa, the bladder only taking 4 oz. Lymph flakes, but no phosphatic deposits, were seen. He was classified as a case of phosphaturia owing to the heavy deposit of phosphate in his urine."

The patient said that since 1914 he had seen sediment in his urine, and had been troubled with frequency of micturition, urgency, and dysuria. In February, 1930, he could hold his water for about two hours. Up to this time he had for many years carried on his occupation of a labourer. He denied ever having had syphilis or gonorrhœa.

ON EXAMINATION.—The patient was of spare build, but otherwise looked well. Both epididymes were somewhat thickened but not adherent to the testes. There was no evidence of urethral discharge. On rectal examination nothing abnormal could be noted. The urine was turbid and contained débris, which was more marked in the second than in the first glass. A detailed report on the urine showed the presence of amorphous and triple phosphate and a moderate number of pus cells and bladder débris entangled in mucus on microscopical examination, while a strong growth of B. coli resulted from cultivation. Investigations for tubercle bacilli were carried out on a number of occasions without revealing the presence of these organisms.

An endeavour to cystoscope the patient was unsatisfactory, as on attempting to fill the bladder the fluid at once returned outside the cystoscope, and no more than one ounce was retained. Under these conditions the only information obtained was that the vesical mucosa was pale and covered with masses of mucoid material. Posterior urethroscopy showed the roof of the posterior urethra to have the same pallor as the bladder.

A plain radiographic examination of the urinary tract showed no evidence of calculus. Further radiograms after the intravenous injection of uroselectan showed no further abnormality in outline of the kidneys or ureters than a very slight degree

of dilatation on the right side, but the poor density of the shadows indicated reduced renal function on both sides. Blood examination showed Wassermann negative, Vernes 0, and resorcin test with an optic density of 95.

OPERATION.—I decided to investigate further by opening the bladder supra-pubically. On doing so, I found a thin-walled organ which had all the appearance of a normal capacity, but presenting a most remarkable appearance (*Fig.* 18). The mucous membrane was uniformly bluish-pink in colour, except on the trigone, where ulceration was present. At the margins of the incised vesical mucosa the thickening of the vesical epithelium was apparent. The ulcerated area roughly occupied the trigone, with several finger-like processes extending beyond these limits, one of them reaching towards and beyond the left ureteric orifice. There was no undermining of the epithelium bordering the ulcer. On the contrary, the edges were slightly rounded from œdema. The thickened epithelium in the vicinity of the internal urinary meatus was somewhat corrugated, doubtless from the contraction of the sphincter.
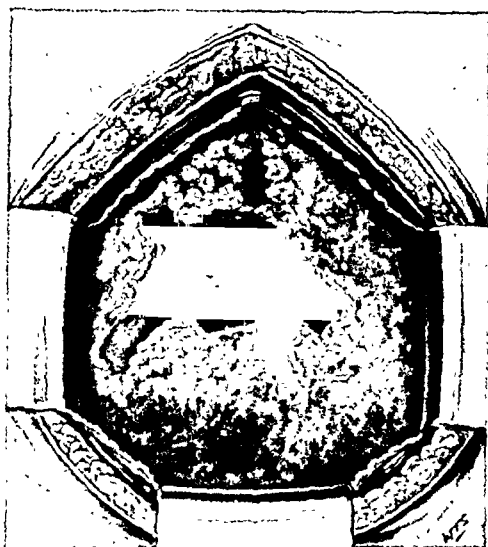

FIG. 18.—View of bladder base through a suprapubic exposure, showing internal urinary meatus, and the extensive ulcer on the trigone.

A striking feature of the mucous membrane was the way the whole surface except the trigone became and remained completely dry after swabbing. Scattered particles of non-adherent phosphatic grit were removed by the same process. The ultimate result was a dry, glistening, bluish-pink appearance, suggesting the new skin formed over a somewhat recent scar.

An attempt was made to catheterize the ureters during this investigation, but the difficult approach to the left orifice made it impossible on this side, while the opposite orifice was so obscured by thickened epithelium that it could not be found.

After removing portions of tissue for microscopical examination from the margin of the ulcer, and from the bladder wall at the site of the vesical incision, the wound was closed round a tube draining the bladder. The microscopic sections both showed that the epithelial lining of the bladder was squamous in type, with keratinization of the superficial layers, a well-marked stratum granulosum, and well-developed and numerous tongues of epithelium projecting deeply into the underlying connective tissue. In places masses of epithelium isolated from the over-lying layer indicated the marked degree of epithelial hyperplasia (*Fig.* 19). The penetration of these cell masses could not be found so deeply as the muscular coat,

and their isolation from the superficial epithelium is readily explained by the manner of cutting of the section with the knife. No area could be identified which would justify the diagnosis of malignancy, but the evidence of marked epithelial activity indicated the tendency towards malignant transformation.

RADIUM TREATMENT.—Up to twelve weeks after the operation the suprapubic fistula had failed to close completely. In consultation with my colleague, Mr. Stanford Cade, it was decided to implant radium under the ulcerated area. Mr. Cade undertook this, and I had the benefit of assisting him. I was able to note that the condition of the ulcer on the trigone was not appreciably altered from that which existed at the previous cystotomy. Four 1·3-mgrm. radium needles were inserted beneath the ulcerated surface and left in position for seven days. The suprapubic fistula was soundly closed in seven weeks, and the patient was discharged from hospital and returned to his work.

FIG. 19.—Microscopic section showing marked hyperplasia of vesical epithelium. (× 50.)

Before he left hospital cystoscopy showed the same appearance of the bladder as before, but with regard to the trigone, although some œdema was noted, no ulceration could now be seen.

SUBSEQUENT PROGRESS.—Nine months later the patient responded to an invitation to report for a further examination. After discharge from hospital he had resumed his manual work, and was still in employment. Although he was a little thin, he had the appearance of fairly good health. On this occasion it was found that the patient's symptoms were practically the same as before I first opened the bladder. It was noted, however, that there was a slight mucopurulent urethral discharge. This apparently had been present for several months. No gonococci were to be found in subsequent bacteriological examination.

In accounting for the discharge one remembered that the leucoplakia had been observed in the posterior urethra on urethroscopy. The urine was still turbid and contained débris. An attempt at cystoscopy was no more successful than on previous occasions, the irrigation fluid being ejected at once outside of the cystoscope.

In this case the outstanding feature was the uniform distribution of the change over the whole of the vesical mucosa except for. the trigone, which

was ulcerated. Microscopically there was a conversion of transitional epithelium into the squamous type.

Usually all stages between the normal and well-developed squamous epithelium can be found if sections are made in different localities. The surface has a layer of varying thickness of cornified epithelium. Instead of a uniform stratum of about four layers of stratified epithelium which normally lines the epithelial surface, there is a considerable depth of squamous cells with down-growing processes extending well into the underlying connective tissue in places. Microscopic sections taken through a portion of tissue where carcinoma has developed indicate the malignant condition as but a further stage of the epithelial change. Therefore the precancerous nature of the leucoplakia seems established. Patch found thirteen cases in the literature in which squamous-celled carcinoma and leucoplakia were found simultaneously in the kidneys, ureters, or bladder. With regard to the bladder in particular, a number of cases have been reported in which the leucoplakia commenced in, and in some cases was confined to, a vesical diverticulum.

The most acceptable theory of the etiology of leucoplakia is that it is a deficiency disease. Two investigators, Wolbach and Howe.[2] found extensive epithelial metaplasia in vitamin A deficiency, which was not confined to, but included, the urinary tract. The changes noted had the features of leucoplakia. More recent experimental work by McCarrison[3] seems to confirm this view. Working with, not only large numbers of rats, but also guinea-pigs, rabbits, pigeons, and monkeys, he has been able to demonstrate the fact that diets with vitamin A deficiency produce amongst other diseases many instances of keratinization of the epithelium of mucous surfaces. This included the bladder, ureters, and renal pelves. The diets he used were composed mainly of cereal grains or cereal products and vegetable fats, with little or no milk, butter, and fresh vegetables. Of all the faulty diets he has used, that composed of white bread, margarine, tea, sugar, jam, preserved meat, and scanty overcooked vegetables proved the most injurious. The significance of this fact is that such a diet is common in poor communities.

Wilson and Du Bois[4] in a post-mortem on a child that died of vitamin A deficiency found widespread keratinization of epithelium in glandular tissue and of mucous surfaces, including the renal pelvis. This is a valuable piece of evidence linking together experimental and clinical findings. Previously two views attempting to explain the etiology commanded attention : one, that the abnormal epithelialization is a result of misplaced embryonal rests of ectoderm ; the other, the theory of metaplasia which attributes the cornification to irritation. The latter possibility seemed to find support from the fact that so many cases were associated with chronic infection.

The obvious difficulty in determining the true relationship between infection and epithelial changes is to know which existed first. If infection is to be considered the cause rather than the effect, the question naturally arises why leucoplakia is so comparatively rare while infection is common. Examination of the bacteriological reports dealing with the infections has thrown no light on the etiology, as a great variety of organisms have been described. But McCarrison has shown that infection is also a common result of deficiency in diet. This evidence therefore considerably weakens the theory

of metaplasia. This investigator maintains that the epithelial changes noted are independent of infection, though this is often superimposed on them, and that they are in the nature of breaches in the body defences against bacterial invasion. He regards vitamin A as an anti-infective agent, and vitamins B, C, and D as having the same qualities, which they exercise in a different way.

Histological examinations of renal pelves not the seat of leucoplakia have revealed many instances in which the tendency towards the condition has been observed, but without giving any clue to the cause. Lavonius[5] microscoped 150 renal pelves and demonstrated 5 cases in which leucoplakia might originate. Leber[6] observed widespread evidence of epithelial proliferation in a girl of four months. The patient had xanthoma of both conjunctivæ, and hyperplasia of the epithelium of the renal pelves. Lubarsch[7] in 160 autopsies found 6 cases with islands of leucoplakia, while Heyman[8] noted small circumscribed areas of cornification in 10 of 20 cases.

The epithelial lining of any part of the urinary tract may be the seat of leucoplakia. With regard to the kidneys and ureters, the disease is not uncommonly bilateral. In hydronephrosis small areas of cornification are sometimes seen. More cases have been reported involving the bladder than the renal pelvis. Hennessey's analysis of 80 cases gave 42 for the former and 24 for the latter. In the remaining cases the lesions were scattered about the urinary tract.

The naked-eye characters of the lesions are most frequently displayed as single or multiple raised grey patches of varying sizes, or there is a uniform hyaline appearance of the mucous membrane of the whole or a portion of the part involved.

With regard to age, Hennessey[9] found in reviewing 74 cases that this varied between 4 months and 71 years, with an average of 38 years. The same writer found an incidence of three males to one female.

The length of time covered by the symptoms varies enormously in reported cases. Kutzmann,[10] in reviewing 67 cases, found it ranged between 3 weeks and 35 years. The symptoms in any given case will be influenced largely by the seat of the disease and the nature of the conditions which accompany it, such as stone, tuberculosis, vesical diverticulum, hydronephrosis, and the degree of infection. No symptoms, therefore, can be put forward as being characteristic of leucoplakia. However, relating to both bladder and kidneys, signs of chronic infection are the commonest features which have been noted. Hæmaturia is frequently noted. Kutzmann reported this symptom in 35 per cent of 67 cases he analysed. In my own case, in which as far as one knows the disease is confined to the bladder and posterior urethra, hæmaturia was one of the first complaints, otherwise the symptoms were continued frequency of micturition, terminal discomfort on micturition, turbidity of the urine, and latterly a slight mucopurulent urethral discharge.

When the disease is confined to the upper urinary tract the true diagnosis will only be made by an inspection of the parts such as is possible with exploration of the kidney, nephrectomy, or at autopsy.

When there is leucoplakia of the bladder, cystoscopy may prove unsatisfactory from the fact that it may be impossible to get the bladder to retain sufficient fluid for this examination to be carried out. In these circumstances

it may be impossible to locate the ureteric orifices. This was the state of affairs in my case, and in several other cases that were reported.

When a view of an area of vesical leucoplakia is obtained, the outstanding features are the absence of visible blood-vessels on the mucosa, which is pale or greyish in aspect. In other cases actual raised pale plaques and areas of ulceration have been seen.

When the upper urinary tract is the seat of leucoplakia, provided that unilateral renal disease has been established, nephrectomy is generally undertaken for the accompanying condition, which usually dominates the clinical picture. If the disease is bilateral, then it is impossible to eradicate it. For the lower urinary tract no curative treatment is known.

Claims have been made by one writer of the benefit of antisyphilitic treatment. These have not been endorsed by others. Radium and electro-coagulation have been helpful as palliative measures.

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PREVASCULAR FEMORAL HERNIA.

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SEVERAL unusual forms of femoral hernia, differing from the ordinary one in some definite anatomical particular, have been described. These are: (1) External, or Hesselbach's, hernia, situated external to the deep epigastric artery and associated with an inguinal hernia ; (2) Hernia through Gimbernat's ligament, or Laugier's hernia ; (3) Pectineal, or Cloquet's, hernia ; (4) Retro-vascular hernia ; (5) Prevascular hernia. All these are rare, and most of them are very rare, but one, the *prevascular hernia*, is probably less uncommon than is usually supposed. This view was expressed in 1924 by L. F. Watson,[1] of Chicago, and gained support from the previous writings of Moschcowitz[2] and Cevario.[3] A number of instances have been reported in the literature, but none, as far as I know, in this country, so that a brief account of my experience may prove of interest.

The usual theory of the origin of femoral hernia attributes it to the presence of a congenital peritoneal diverticulum on the inner side of the femoral vein. This diverticulum may be supposed to be formed by being carried out with the vessels as they grow down into the limb bud of the embryo. Usually the diverticulum is shallow, but is increased by intra-abdominal pressure so that it grows in the line of least resistance—that is, down the femoral canal and into the thigh through the saphenous opening. This is the state of affairs in the vast majority of femoral hernias, the fundus of the sac being in Scarpa's triangle, and the neck in the femoral canal. Now if this theory of the common origin of femoral hernia is correct, it might be supposed that very occasionally a much longer peritoneal diverticulum might be dragged out with the growing vessels, and, accompanying them down the thigh, be found within the femoral sheath in front of the vessels. The fundus of the hernial sac would then be far below the level of the saphenous opening, and would never tend to emerge through it. This would in fact constitute the condition of prevascular femoral hernia, an example of which has come under my observation as follows :—

Case 1.—A young labourer, age 23, attended at the City of London Truss Society on Nov. 25, 1924. He complained of a lump in the right groin, which had given him occasional pain and one attack of severe pain and retching. On examination he was found to have a right femoral hernia, containing loculated fluid in the fundus, so that the hernia was clinically ‘irreducible’. He had never noticed anything abnormal on the left side, but in the course of my routine examination I detected signs of a reducible hernia in the centre of Scarpa's triangle immediately in the line of the femoral vessels. Nothing abnormal was to be seen, but an impulse could be felt when he coughed, and reduction of the very slight swelling then formed was accompanied by the sensation usually produced by the slipping back of an epiplocele.

An operation was performed on Dec. 5, and on the right side a loculated femoral hernia was exposed in the usual position, the neck lying in the saphenous opening and femoral canal.   On the left side no hernial sac could be found in this position, but the femoral sheath appeared wider than usual.   A vertical incision was therefore made in the centre of the sheath, and a long narrow peritoneal sac was exposed as it lay on the vessels inside the sheath.   The fundus was dragged up through the incision in the sheath and the appearance was then as shown in *Fig.* 20.   The sac was 6 cm. in length, so that it extended some way below the level of the saphenous opening, and was 2 cm. wide at its origin.   It was found to be empty when opened, and it was removed by the inguinal route in the ordinary way.   The patient was
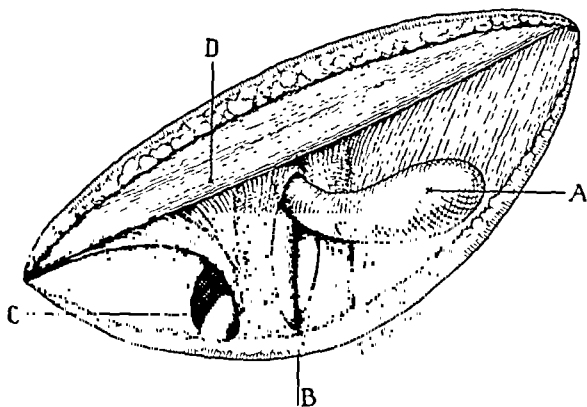

FIG. 20.—*Case* 1.   Dissection of congenital prevascular femoral hernia.   A, Fundus of sac ;
B, Femoral sheath ;   C, Saphenous opening ;   D, Inguinal ligament.

young and muscular, so that the manœuvre of approximating the lower edge of the internal oblique fascia or the conjoint tendon to the pectineal fascia would have produced great tension in these structures.   A plastic operation, such as I have described elsewhere,[4] was therefore carried out on both sides, a flap of the internal oblique layer of the anterior rectus sheath being stitched to the ileo-pectineal line. On the left side the vessels were retracted outwards further than usual, and four sutures were used, so that the vessels when released came into very close contact with the outer edge of the flap, which was also sutured to the inguinal ligament in front of the vessels.   The patient made a normal recovery, and has been doing heavy work ever since without any sign of recurrence on either side (7 years).

Moschcowitz claims to have described the first case " operated upon and studied *in vivo* " in 1912, and the above description agrees with the condition found by him.   It is noticeable that in this instance the congenital origin of the hernia seems to be unquestionable, since the fundus, as shown in the drawing, is appreciably wider than the neck, which would scarcely be the case had the hernia been acquired.   It offers therefore a striking contrast to another instance of prevascular femoral hernia which I believe had been acquired.

Case 2.—A man, age 60, was seen at the City of London Truss Society in March, 1931.   He complained of a hernia on the right side, which again was in Scarpa's triangle immediately over the femoral vessels.   In this patient, however, the hernia was conspicuous when he was standing, and formed a roughly triangular pouch with its base upwards, as shown in *Fig.* 21.   His tissues exhibited a well-marked degree of senile laxity, and the hernial sac appeared to have formed in the upper part of

the femoral sheath as the connective tissues sagged, the 'neck', or opening, being much wider than the fundus. A lesser degree of the same condition was noticed on the left side. The patient declined to consider operative treatment and was given a truss, so that the anatomical state of the parts could not be verified by dissection, though I was quite satisfied that the condition was as I have described.

FIG. 21.—Acquired bilateral prevascular femoral hernia.

The distinction between congenital and acquired prevascular femoral hernia has not, as far as I am aware, been clearly made before. Minor degrees of the acquired condition are probably not so very uncommon, though among many thousand patients seen by me at the City of London Truss Society during the last twelve years none has been so well marked as that illustrated in the photograph of *Case* 2. It is probable that this condition would not be amenable to operation, though the congenital variety is curable by a suitable operation as demonstrated in the first case.

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RECONSTRUCTION OF FOREARM AFTER LOSS OF RADIUS.

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WILLIAM A., age 6½ years, fell in the school playground one day in February, 1921, injuring his right forearm. It is not known if he sustained an open fracture of the radius and so received a direct infection of the bone, but certain it is that an osteomyelitis of the radius ensued, and on July 15 an X-ray examination revealed necrosis of the shaft, and some sequestra were removed through sinuses. By Aug. 28, the patient being still under treatment at the School Clinic, the whole shaft of the radius lay exposed in the depths of an open suppurating wound. He was then referred to Dr. Holland, of Swindon, who transferred the case on Sept. 10 to the Bristol General Hospital, where on Oct. 15 a large sequestrum, comprising the greater part of the shaft of the right radius, was removed.

The case was referred to me on Aug. 14, 1922. There were still several discharging sinuses, which finally healed by Nov. 3. There remained only part of the lower epiphysis and a long slender portion of the upper end of the bone, which appeared from an X-ray to be sclerosed and unpromising as a bed for the reception of a bone-graft. The large gap was filled by a mass of dense fibrous tissue, and most of the overlying skin of the back of the forearm was converted into scar tissue. The hand was very helpless, being deflected to the radial side, partly by lack of support of the hand by the defective radius, and partly by contraction of the dense scar tissue (*Fig.* 22).

FIG. 22 —Condition of right hand and forearm on Nov. 3, 1922, before operation.

As a prelude to the transplanting of a graft from the rib, a large tubular skin-graft was taken from the abdomen after removing the scar tissue between the ends of the radius, and this was soundly healed in position on the back of the forearm by Dec. 19, 1922.

When the time for transplanting the bone-graft arrived (April 19, 1923), the reluctant patient, now 9 years old, protested vigorously and did not again present himself at the hospital until early in 1925. The forearm was

again X-rayed by Dr. Harrison Orton, who reported that the poor quality of the upper fragment precluded any likelihood of a successful bone-graft being achieved. Mr. Hey Groves,[1] moreover, had described and illustrated a somewhat similar case, in which two successive attempts at bone-grafting had failed. On these considerations grafting was abandoned and it was decided to adopt a device similar to that employed by Mr. Hey Groves, in order to lessen the serious disability, more especially as the parents (and the boy) were wearied of operations and desired finality.

OPERATION (May 5, 1925).—A rectangular incision through the skin was made across the line of the wrist-joint and up the forearm along the ulnar border for about 3 in. (*Fig.* 23, A B C). The triangular flap so marked out was raised, the tendons retracted, and the bones exposed.

In the case previously referred to (the only other case I have been able to find recorded), the ulna was cut through close to the distal radio-ulnar joint and the lower end of the ulna with its styloid process, was removed. The upper

FIG. 23.—Diagram showing bones before operation and line of incision (A B C) through the skin.

fragment was then trimmed to a conical shape and fixed into a hollow scooped out of the upper surface of the distal portion of the radius. In my case a variant of this method was adopted. Out of the upper surface of the lower fragment of the radius a wedge-shaped piece was cut. The
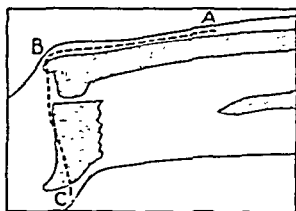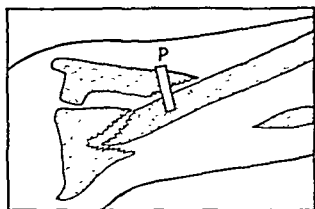

FIG. 24.—Diagram showing position of bones after completion of operation. P. Bone-peg.

ulna was then divided obliquely from above downwards and outwards some 2 in. above the level of the wrist-joint (*Fig.* 24); the pointed lower end of the upper fragment fitted into the V-shaped cavity in the radial epiphysis, whilst the lower fragment of the severed ulna was fixed against the shaft of the ulna by a bone-peg, square in section (P), thus acting as a buttress. In this way the integrity of the wrist-joint was not violated; the resulting joint is perhaps more stable and possibly more mobile.

In *Fig.* 25 the condition of the bones of the forearm at the present time (March 8, 1932) is seen, and *Fig.* 26 shows the appearance of the limb. The ulna measured from olecranon to styloid process is 7 in. long, whereas that of the other forearm measures 11 in. It is evident that little growth has taken place at the epiphysial line, and this confirms the opinion that an attempt at bone-grafting would probably fail; even if successful, there would have resulted considerable disparity in length between the radius and ulna.

The patient now has a strong forearm with a good grip, free flexion and extension, and a small degree of pronation and supination, so that he is able to lift heavy weights and do laborious manual work. In the X-ray photograph (*Fig.* 25) it will be seen how effectively the buttress strengthens the joint.

COMMENTS.

1. The loss of the shaft of the right radius by necrosis caused a major disability for which the only solution seems to be that adopted in this case.

FIG. 25.—Condition of bones of forearm in March, 1932.

2. The injury was sustained in a school playground and the case remained under the care of the School Clinic for about seven months. This illustrates the wide latitude assumed by Medical Officers of Health nowadays in the treatment of serious injuries.

3. The serious disability tended to increase with the approach of adolescence. The successful grafting of the radius would have failed to correct this, since the capacity for growth in the distal radial epiphysis would have prevented the radius keeping pace with the ulna. The method adopted would appear to be the only practical solution of the problem.

4. The shaft of the ulna has increased in thickness to about double the size of the normal bone.

5. Using the lower portion of the ulna as a buttress, instead of removing it as was done in the case previously recorded, seems likely to give greater stability to the wrist-joint.

6. The rarefaction of the carpal and metacarpal bones seen in an X-ray photograph taken in 1925 has disappeared by March, 1932, and is evidence of the increased use of the limbs.

FIG. 26.—Appearance of limb in March, 1932.

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A NOTE ON THE MOVEMENTS OF THE SHOULDER-JOINT.

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DURING a recent investigation into the mechanism of the movements occurring at the shoulder-joint some new facts about these movements became apparent and appear to be worthy of record. No reference could be found in any of the anatomical text-books or journals to some of the observations which appear to have considerable practical importance in connection with common injuries and diseases of the joint.

The movements of the shoulder-joint, as described in recognized text-books of anatomy, are : flexion and extension, abduction and adduction, circumduction, and finally rotation. By flexion is meant a carrying of the arm forwards and then upwards, the arm being parallel to the median plane of the body at all stages of the movement. By abduction is meant a carry-ing of the arm outwards and then upwards, the arm being first parallel to the median plane, then at right angles to it, and finally parallel to it once more. Circumduction and rotation do not need any explanation. In dealing with the actual carrying out of these movements the text-books emphasize the important part played by rotation of the scapula and elevation of the clavicle, especially in flexion and abduction of the arm. Both these move-ments are carried out not solely at the humero-scapular joint, but are due to a considerable extent to movements at the sterno-clavicular and acromio-clavicular joints. This applies especially to the later stages of the movements of full flexion and full abduction. It is often stated, as, for example, in *Quain's Anatomy*[1] and many other text-books, that the amount of abduction which can take place at the humero-scapular joint is limited to carrying the arm outwards till it is at 64° to the median plane, abduction beyond this point being effected entirely by rotation of the scapula. It has been shown, however, that some rotation of the scapula occurs from the very beginning of either abduction or flexion, and some movement at the humero-scapular joint continues till the arm is fully upright.[2]

From the descriptions given in the text-books, therefore, it would appear that flexion of the arm and abduction of the arm are very different move-ments. In the case of abduction the arm is carried out till it is at right angles to the median plane. The head of the humerus has therefore been rotated round an axis passing horizontally from before backwards, the greater tuberosity of the humerus has been brought almost against the acromion of the scapula, and the articular surface of the head of the humerus looks down-wards, slightly medially, and slightly backwards. Any further abduction that may be carried out is due mainly to rotation of the scapula, but, as already stated, some further movement occurs at the humero-scapular joint.

In full abduction, therefore, the articular surface of the head of the humerus must look almost directly laterally, and be only very slightly in contact with the articular surface of the glenoid cavity of the scapula. In the case of flexion, on the other hand, the head of the humerus is rotated round an axis passing in a transverse horizontal direction ; the articular surface of the head of the humerus, therefore, remains in contact with the surface of the glenoid cavity, and in full flexion the head of the humerus looks medially and slightly forwards. Of course in flexion, as in abduction, rotation of the scapula and elevation of the clavicle take place.

The above, I think, is a fair account of the movements of this joint as given in current anatomical literature. With the movements occurring at the sterno-clavicular and acromio-clavicular joints this paper is not concerned, as the text-book descriptions of these movements appear to be complete.

Now it is obvious that we can carry our arms straight up over our heads by two methods, either by flexion or by abduction. From the text-book description of these movements we should infer that the end-result in each case would be different. Observation, however, will show us that the end-results in both cases are identical. If we carry our arms straight upwards it makes no difference, as regards the final position of the bones, whether we do so by flexion or abduction. This fact can easily be verified. With the arm hanging downwards by the side the lateral epicondyle of the humerus is directed forwards and laterally. If the arm is now carried vertically upwards by flexing the limb it will be found that the lateral epicondyle is directed backwards and slightly laterally—in other words, the head of the humerus has, as already stated, rotated round an axis passing horizontally from side to side. If now the arm is again placed by the side and then carried vertically upwards by abduction it will be found that the lateral epicondyle is in exactly the same position as it was when the arm was carried upwards by flexion. So obviously in the case of abduction something more than rotation of the head of the humerus round a horizontal antero-posterior axis has taken place, and the description of abduction in the anatomical text-books appears to be incomplete.

If, once again, the arm is placed hanging down by the side of the body and then carried slowly upwards by abduction, while the position of the lateral epicondyle of the humerus is noted at all stages of the movement, it will be found that, as the arm is abducted, the humerus is rotated laterally round its own long axis. This lateral rotation of the humerus during abduction of the arm is the fact to which I desire to direct attention, for, as far as I can find, it has not been described or noticed in any account of the movements of the shoulder-joint.*

Another method of demonstrating the lateral rotation of the humerus during abduction of the arm is as follows : Place both arms hanging down beside the body, flex the elbow-joint till the forearm is at right angles to

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* Since this paper was written my attention has been drawn to *Applied Anatomy and Kinesiology* by Bowen and McKenzie, published by Lea & Febiger, of Philadelphia and New York, 1919. On p. 101 of this work there is a passing reference to the fact that the humerus rotates laterally during abduction of the arm. No details of the movement are given.

the arm so that the forearm points directly forwards, then abduct the arm, keeping the elbow flexed and the forearm pointing forwards all the time. It will be found that the arms can only be abducted to a little over the horizontal position even though the scapula itself has been raised as much as possible. The reason for this limitation of abduction is that, by keeping the forearm pointing forwards while the elbow-joint is flexed to a right angle,
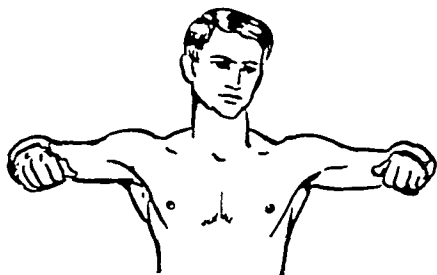

FIG. 27.—Arms abducted as far as possible while lateral rotation of the humerus is prevented by elbow being flexed to a right angle and forearm being kept pointing forwards.
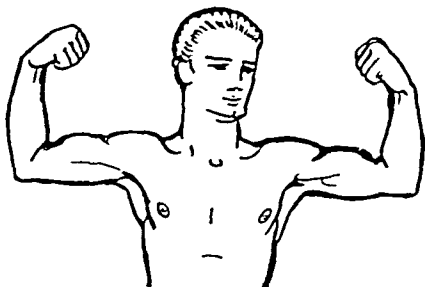
FIG. 28.—Arms in same position as in *Fig.* 27, but humeri turned laterally by turning forearms so that they point upwards.

we have stopped, or at least largely diminished, the lateral rotation of the humerus. If we now, still keeping the elbow flexed, rotate the humerus laterally so that the forearm points upwards instead of forwards we shall find that the arm can be fully abducted till it is directly vertical. (*Figs.* 27–29.) In applying this test it is better to use both arms at the same time, otherwise abduction of one arm may be assisted by tilting of the thorax and the lateral rotation of the humerus obscured. Again, it is obvious that with the arm fully abducted we can flex the forearm inwards over the head, but we cannot flex it outwards ; likewise with the arm hanging at rest beside the body we can flex the forearm inwards but not outwards. Of course, owing to rotation of the humerus, we can vary these positions to a certain degree, but still the general direction in which the forearm can be flexed is inwards towards the body, whether the arm is hanging by the side or is vertically upwards in full

FIG. 29.—Showing full abduction, rendered possible by lateral rotation of humeri.

abduction. Here again it is clear that if rotation of the humerus during abduction had not taken place, then, when the arm is fully abducted, we should be able to flex the arm outwards.
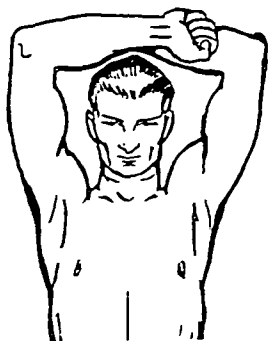
The plates published by Lockhart[2] show this lateral rotation of the humerus during abduction very clearly, but the fact of the rotation was apparently not observed by him. He draws attention to the fact that abduction of the arm in its later stages—that is, from the horizontal position to when it is vertically upright—is not accomplished wholly by rotation of the

scapula but is attended by considerable movement at the humero-scapular joint ; but he does not remark that a very conspicuous part of this movement consists of lateral rotation of the humerus. Fick[3] published a very full account of the movement of the joints, but I cannot find that he noticed this feature of abduction of the arm. I need not enumerate the modern text-books of anatomy that have been searched in vain for any reference to this feature.

This rotation of the humerus during abduction also serves to explain the mechanism of dislocation of the shoulder-joint. The majority of these dislocations appear to be due to sudden hyperabduction of the arm while lateral rotation of the humerus is prevented, either by the suddenness of the movement or by contraction of the muscles passing from the thorax to the anterior aspect of the humerus. This latter contingency arises when, with the forearm flexed to a right angle with the arm, a man falls sideways and the volar aspect of the hand and forearm comes in contact with the ground. In the effort to save himself he contracts all the muscles passing from the thorax to the arm, including the teres major, latissimus dorsi, and pectoralis major, all of which are inserted into the anterior aspect of the humerus, and, while the impact of the fall forces the arm up into the position of full abduction, the contraction of these muscles prevents lateral rotation of the humerus.

Taking into account the fact that so-called abduction of the arm is a very complicated movement involving (1) true abduction—that is, a carrying of the arm away from the body in a lateral direction, (2) lateral rotation of the humerus, and (3) rotation of the scapula and elevation of the clavicle, we can see how it comes about that the position of the bones at the end of abduction is exactly the same as if the arm was carried to the vertically upright position by flexion. It would also appear that great care should be exercised in examining cases of limitation of movement of the shoulder-joint, for, if this is not done, many such cases may be wrongly diagnosed as impairment of the power to abduct, whereas the real impairment is the inability to rotate. In fact, it is also possible that some of the splints and arrangements used in cases of injury in the vicinity of the shoulder-joint may be ill advised because they do not allow for this lateral rotation ; and, from what has been written above, it is apparent that if the power to rotate the humerus is lost, abduction of the arm must be gravely affected. In this respect it is of interest to note that Jones and Lovett and other orthopædic surgeons advocate putting up all such injuries with the arm fully rotated laterally so that the forearm points upwards when the elbow is flexed to a right angle.[4] They thus recognize that this position gives the best end-results, but they apparently do not recognize the reason for this fact.

In abducting the arm the muscles which contract are the supraspinatus, deltoideus, and trapezius. Other muscles in the neighbourhood are relaxed. The lateral rotation of the humerus during abduction of the arm is apparently brought about by the muscles which are contracting, and of these the most important in producing the rotation of the humerus are the posterior fibres of the deltoideus which take origin from the lower lip of the crest of the spine of the scapula and are inserted into the deltoid eminence on the humerus. The rotation is aided by the fact that the deltoid eminence of

the humerus is slightly more on the anterior than the posterior aspect of the bone. The supraspinatus may help in procuring this rotation, as it arises slightly behind the shoulder-joint and is inserted directly lateral to it. But in the latter half of the movement of abduction the rotation of the humerus is mainly due to the position of the scapula and the outline of the lateral edge and under surface of the acromion and coraco-acromial ligament. These two structures form an arch over the glenoid cavity of the scapula, but it should be noted that this arch is nearer to the glenoid cavity in front than it is behind. It should also be noted that though some rotation of the humerus takes place during abduction of the arm from the side to the horizontal position, most of it occurs during the latter half of abduction when the arm is carried from the horizontal position to the vertically upright one, and it is during this later stage that the greater tuberosity of the humerus comes in contact with the lateral edge and under surface of the acromion and coraco-acromial ligament. As the arch formed by these two structures is farther from the glenoid cavity behind than it is in front, the greater tuberosity of the humerus slides backwards when it comes in contact with them and thus rotates the humerus laterally. Finally, the greater tuberosity of the humerus is directed straight backwards and the shaft of the humerus occupies the interval between the acromion and the coracoid process. The infraspinatus and teres minor might from their position appear to help in rotating the humerus laterally during abduction, but they are relaxed in abducting the arm, and in addition their tendency to rotate the humerus laterally is outbalanced by the tendency of the pectoralis major, teres major, latissimus dorsi, and subscapularis to rotate the humerus medially. It is evident, therefore, that all the muscles which are relaxed in the act of abduction have little influence on the rotation of the humerus.

In abducting the arm we contract one set of muscles and stretch another, and if we use the arm as a tensile organ and hang by it from a beam it is clear that the tensile strain of the body weight will be transmitted through the set of muscles that have been stretched. These stretched muscles are the pectoralis major, the teres major, and the latissimus dorsi, all of which are inserted into the anterior aspect of the humerus. But if the humerus is rotated laterally during abduction it would appear that all these muscles have undergone a good deal of torsion, and we should expect that if a man was hanging by one arm his body would swing round till the torsion was undone. This is what actually happens. Suspended by one arm the body will not come to rest till it has rotated round the humerus so as to undo the lateral rotation of this bone and the torsion of these muscles. The arm is then in a position of true abduction combined with considerable tilting of the thorax. This is an experiment that anyone can try on himself. Of course if the arm is carried up by flexion similar torsion of the muscles occurs.

SUMMARY.

Abduction of the arm is a complicated movement consisting of true abduction of the humerus at the humero-scapular joint combined with rotation of the scapula and elevation of the clavicle. But, in addition to these

factors, abduction of the arm is always accompanied by lateral rotation of the humerus to an extent of over 90°. This rotation takes place for the most part during the later stages of abduction when the arm is raised from the horizontal position until it is vertically upright, but a certain amount of it occurs during the first half of abduction. It is on account of this lateral rotation of the humerus during abduction that the position of the humerus at the end of full abduction is exactly the same as it is at the end of full flexion. This lateral rotation of the humerus during abduction of the arm has not, as far as I can find, been described in any publication previous to this paper.

I desire to thank Miss O'Brien for drawing the illustrations for this paper.

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SOME EXPERIENCES WITH SYMPATHETIC GANGLIONECTOMY.

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THE sympathetic nervous system is rapidly coming to the forefront of surgical interest, and both Lord Moynihan, in this country, and W. J. Mayo, in America, have expressed the opinion that much in the future development of surgery will lie in the direction of the autonomic nervous system. A more complete understanding of its anatomy and physiology is still wanting, though much has already emerged from the abundance of research which is being pursued in most countries at the present time. It is a matter of no small satisfaction to realize that the pioneer work in this sphere came from the British school of physiology. the names of Gaskell and Langley being pre-eminent ; indeed, Albert Kuntz,[1] in introducing his book, *The Autonomic Nervous System*, a complete résumé of all our knowledge of this subject up to 1929, says of the latter, " the term autonomic nervous system ought to be used in recognition of Langley's great contribution to our knowledge."

The autonomic nervous system is divisible into two antagonistic parts— the parasympathetic and the sympathetic, and it is to the latter that surgical procedures have been directed. No attempt will be made here to discuss all that is known of the physiology of the whole autonomic system. though a little must be said of those recognized functions of the sympathetic part of the system which surgeons have attempted to modify in some way or other by operative interference ; it will be evident. too, how these surgical experiences have stimulated further research and have led to fresh knowledge.

It has long been known that stimulation of the nerve-fibres lying within the adventitia of an artery leads to spasm of that artery, and Leriche, seeing the practical application of this fact, evolved his operation of peri-arterial sympathectomy. This he used for vasospastic conditions of the legs and arms, particularly Raynaud's disease and Buerger's disease (thrombo-angiitis obliterans). He also applied it to such maladies as chronic varicose or traumatic ulceration of the legs, delayed union of fractures, and even to traumatic tuberculosis of joints, conditions, he argued, wherein any improvement in circulation would seem to promise a stimulus to the sluggish processes of healing. This vasodilatation—for that is what the operation was calculated to produce—is found to be of temporary duration only,[2] lasting about five to six weeks, after which time the arteries would appear to regain their normal tone, so that. though of considerable help in the more local conditions such as the three last mentioned. in which a fuller blood-supply of a few weeks' duration is sufficient to initiate a more vigorous healing reaction, the results are disappointing in the more generalized states such as Raynaud's and Buerger's

diseases, in which the improvement in circulation needs to be lasting before permanent alleviation can be expected; the conception was good but the anatomy was incompletely understood.

At about the same time as Leriche was studying these problems of vaso-spastic diseases other workers were attempting to discover some procedure by which the pain of angina pectoris could be permanently controlled. The names associated with these researches were Jaboulay, Jonnesco, and Danielo-polu in Europe, and Coffey, Brown, and Cutler in America. They attacked different parts of the cervical sympathetic chain on the left side on the assump-tion that the pain of angina is due to spasm of the coronary arteries and aorta and that the impulses are conveyed through the afferent components of the sympathetic cardiac nerves. The surgical treatment of angina pectoris has never been popular with British cardiologists, as they consider the pain a useful danger signal and the operations suggested too severe for patients suffering from cardiac lesions. However, as with the problems of vasospastic diseases, this work has stimulated further research.

Quite a different aspect of the sympathetic part of the autonomic nervous system has received attention in Australia, and in 1923 Hunter published his brilliant researches on the dual innervation of the voluntary muscles, and Royle described his equally brilliantly conceived surgical approach to certain spastic conditions of the muscles, based on these researches. He severed the grey rami communicantes of the cervical or lumbar ganglia, according to whether the spasticity involved the arms or legs respectively, with the idea of cutting out the overactive plastic component of muscular tone. His patients, for the most part, were wounded soldiers with spinal injuries—patients, in other words, who formerly had been normal in body and mind, and who, therefore, would co-operate intelligently and enthusiastically in the re-educative muscular exercises which are almost the most important part of treatment. Royle reported many successes, but the operation has not been so encouraging in the hands of others; and Hunter's researches have as yet remained unconfirmed. In civil practice so many of these cases occur in children who have sustained birth injuries, whose mentality is defective, and whose powers of co-operation are distinctly poor. Probably this fact accounts for the failures experienced by many surgeons who have attempted to deal with spastic diplegias. In this country the most that is attempted from a surgical point of view is some modification of Stoffel's operation, and then only if the child exhibits considerable intelligence. Nevertheless, the work of Hunter and Royle aroused universal interest, and observers realized that the varying results of all these different operations were due to an inadequate knowledge of the anatomy of the sympathetic system.

I do not propose to recount in detail the more recent anatomical researches into this problem; suffice it to say that the work of Hovelacque, Delmas, Latarjet, and Langley in Europe, and of Kuntz, Ranson, and Learmonth in America, has demonstrated the following facts :—

1. The nerve-supply to the arteries of the limbs is somatic, not visceral— that is, the nerves lying within the adventitia of the main artery as it enters the limb do not comprise the total nerve-supply of the entire arterial tree distal to this point; there is a constant accession of fibres derived from the

nerves to the voluntary muscles as one passes distally. From a practical point of view it can be said that the nerves lying within the adventitia of the femoral artery just distal to Poupart's ligament supply the artery and its branches as far as the level of the knee and no farther: everything below this derives its nerve-supply from fibres entering the vessels at lower levels. e.g.. a branch from the nerve to the popliteus can always be found to enter the anterior and posterior tibial arteries. In like manner fibres within the adventitia of the third part of the axillary artery supply the tree only so far as the elbow. It is therefore obvious that in order to denervate the whole arterial tree of a limb one must attack the source.

2. Complete sympathetic denervation of the vessels of the lower extremity can only be achieved by removal of the 2nd. 3rd. and 4th lumbar ganglia with the intervening chain on the appropriate side. Complete sympathetic denervation of the vessels of the upper extremities demands extirpation of the inferior cervical ganglion. the 1st and 2nd dorsal ganglia, and the intervening chain. The 2nd dorsal ganglion is of importance. as in over 20 per cent of cases a large branch is found to pass from it into the brachial plexus (Kuntz). Neglect of this may lead to disappointing results.

No account. however brief, would be complete without mentioning the very convincing experiments carried out by Horton and others at the Mayo Clinic. A number of dogs were taken and in one series a peri-arterial sympathectomy was performed in Scarpa's triangle on one side ; in a second series the 2nd. 3rd, and 4th lumbar ganglia and intervening chain were removed on the same side as in the first series ; a third series was taken as a control. It was arranged that the second part of the experiment should be done by a separate group of workers. who were not informed as to the exact details of what had been done in each animal ; and in order to make the investigation more perfect the same incisions were made in each dog. Thus, in the first series, although the operation was performed through an incision in the upper part of the thigh, a second superficial mid-line abdominal incision was also made ; in the second series, although the operation was performed through a mid-line abdominal incision. a second superficial incision was made in the upper part of the thigh ; and in the third series two similar but superficial incisions were made as in the other two series. The experiments were, therefore, perfectly controlled. The second stage of the experiment was carried out about six to eight weeks later, and consisted of opening the abdomen of each anæsthetized dog and injecting mercury at systolic pressure into the abdominal aorta. X-ray pictures of the lower extremities were then taken before death. In the first series the pictures showed dilatation of the arterial tree on the side of operation as far as the level of the knee but no farther; the second series showed dilatation of the whole tree. right down to the tips of the toes, as compared with the untouched side ; the third series showed no difference. The results fulfilled exactly the anticipations of the first group of workers.

3. The circular fibres of the sigmoid colon are supplied by a definite branch of the presacral nerve of Hovelacque, known as the 'lumbar colonic nerve' ; this has recently been confirmed by Learmonth and Markowitz.[3] Excision of the presacral nerve, which is really a plexus, releases the

sympathetic control of the sigmoid, and has been used for certain cases of spastic colon, of which one case is recounted below.

The Conditions for which the Operations have been Advocated.—Experience has shown that the following conditions are amenable to treatment by sympathetic ganglionectomy, but of course all other forms of treatment should be tried before recourse is had to these operations.

1. *Raynaud's Disease.*—This disease, excepting in its most advanced stages, would seem to be due entirely to overaction of the sympathetic nerve-supply ; at any rate, release from sympathetic control checks the arteriolar spasms and thereby leads to a cure.

2. *Thrombo-angiitis Obliterans.*—In this disease there are two factors at work—namely, the inflammatory lesion of the vessels resulting in narrowing of the lumen, and a superimposed spasm. If this latter factor is of sufficient degree, the operation of sympathectomy will result in great improvement— even to checking a threatened gangrene. Whether or not a case is suitable for operation is ascertained in the following manner. A laboratory thermo-meter is bandaged to the affected limb in such a manner that the bulb lies between, and in contact with, two fingers or toes, as the case may be. The temperature is noted, and that of the mouth is also taken. An intravenous injection of 50 millions T.A.B. is then given and the highest subsequent temperatures in both mouth and limb are recorded. The temperature of the limb before the injection may be little above room temperature, and any rise during the fever stage is due to vasodilatation consequent on relaxation of sympathetic tone. Brown, at the Mayo Clinic, has found from experience that if the difference in degrees between the rise of temperature in the mouth and that of the limb is three times the rise in the mouth, then the case is suitable for operation. Thus, if the rise of temperature in the mouth be 3° F. and that in the limb be 16° F., the index becomes $\dfrac{16 - 3}{3} = 4\cdot3$, and the patient would receive great benefit. If the index is below 3 it indicates that the thrombosis is responsible for the trophic changes to a much greater degree than the spasm. In Raynaud's disease the index may be as high as 10 to 20.

3. *Certain Cases of Severe Constipation* due to spasm of the colon are much benefited by removal of the presacral nerve, since the sympathetic not only innervates the circular fibres and sphincters but also inhibits peristalsis.

4. *A Miscellany of Cases exhibiting Minor Trophic Changes,* wherein are present signs of sympathetic overaction in the form of coldness, blueness, clamminess, shininess of the skin, etc. Of such conditions may be mentioned causalgia of amputation stumps, some types of scleroderma, indolent ulcera-tions. In this category should be included also certain types of peri-arthritis, and although the writer has had no personal cases so far, yet from his experience as assistant to Dr. A. W. Adson at the Mayo Clinic he can speak favourably of this method of treatment in carefully selected cases. The necessary features of a suitable case are that there should be no bone changes, that there should be evidence of arterial spasm, and that the joints affected should be peripheral ones. After all, sympathectomy in these circumstances establishes permanently that which many forms of conservative treatment such as radiant heat, diathermy, whirlpool baths, etc., merely succeed in

producing temporarily—namely, vasodilatation; but unless there is quite definite evidence of sympathetic overaction of a degree sufficient to give an index of 3 or over in Brown's test, nothing but disappointment will ensue and the method of treatment will be brought into disrepute.

5. *Certain Types of Pain*, thought to be due to vascular spasm, though not accompanied by indisputable evidence of sympatheticotonia. The anatomical basis for operation in these cases is not entirely established, but some authorities maintain that afferent fibres from the arteries carry painful sensations up to the brain and that sympathetic ganglionectomy, by severing the pathway, will relieve the pain. Certainly, the pain of Raynaud's and Buerger's diseases is abolished by the procedure, but in these conditions the spasms are of such severity that trophic changes develop, whereas in the cases under the present heading the nutrition of the part affected would not appear to be impaired. One case of intermittent claudication is reported below, and, as will be seen, the result was disappointing. Leriche and Bornard have used different forms of retroperitoneal sympathectomy for the type of dysmenorrhœa said to be due to fibrocystic ovaries and have reported successes, but the writer has no experience in this field. As already said the treatment of the pain of angina pectoris by any form of operation is viewed with disfavour in this country.

Choice of Cases.—The operations of dorso-cervical sympathetic ganglionectomy and of lumbar sympathetic ganglionectomy are more severe procedures than Leriche's peri-arterial sympathectomy, and in elderly, enfeebled, toxic patients should not be attempted; the lumbar operation is contra-indicated in obese subjects. In such cases a peri-arterial sympathectomy might, with advantage, be undertaken; but, as already pointed out, it is anatomically incomplete, and when possible the full sympathectomy should be performed.

Operations.—Both operations have been illustrated and described in detail by Adson[4, 5, 6, 7] on more than one occasion, and except for minor differences in technique the following cases were performed by his method, the essential points of which are briefly as follows. The operation for the removal of the inferior cervical and 1st and 2nd dorsal ganglia is based on Henry's exposure of the cervico-dorsal ganglion. The transverse process of the first or second dorsal vertebra and the head and neck of the corresponding rib are removed, and the ganglia are found lying in front, on the pleura. The inferior cervical ganglion lies on the neck of the 1st rib, and the 1st dorsal ganglion is often fused with it. The 2nd dorsal ganglion lies in front of the head of the 2nd rib. The grey and white rami communicantes are seen to enter and leave the ganglia in radiating fashion, and the intervening chain is as thick as the radial nerve, though this is variable. Sometimes, if there has been old apical inflammation, the ganglia and chain are bound by adhesions. If both sides are to be treated, they can be reached by a single dorsal mid-line incision; if only one side requires operation, the incision is made 1 in. lateral to the mid-line. The patient is placed in the laminectomy position for the operation, and an illuminated spatula is essential for the deeper dissection. The dead space resulting from the removal of bone should be obliterated so far as is possible. The patient should be nursed on the back with a pillow between the shoulders.

The 2nd, 3rd, and 4th lumbar ganglia lie on the sides of the bodies of the corresponding vertebræ. They are behind the vena cava on the right side, and the aorta on the left. If it is wished to remove the chain of ganglia on both sides, the operation is best performed through a mid-line abdominal incision with the patient in the Trendelenburg position. On the right side the ganglia are approached through an incision through the posterior abdominal peritoneum, internal to the cæcum and below the root of the mesentery. On the left side it is necessary to incise the peritoneum lateral to the descending colon and to reflect the colon towards the mid-line. Again, an illuminated spatula or a head-light is essential. If it is intended to attack one side only, this can be done retroperitoneally through a lumbar incision as for exposure of the ureter.

The presacral nerve lies in front of the 5th lumbar vertebra in the angle of the bifurcation of the aorta, and the lumbar-colonic nerve runs with the inferior mesenteric artery : both are most satisfactorily removed trans-peritoneally.

CASE REPORTS.

The following are a few examples of cases treated by one or other of these operations. In each case many forms of conservative treatment had been given first but without benefit. The writer feels that sufficient time has elapsed in each case since operation to merit the result being considered satisfactory. They were reviewed in April, 1932, and each one was found to have maintained the original improvement.

Case 1.—J. S., age 21, female. Hairdresser.

Admitted to hospital in February, 1930, complaining of increasing numbness and tingling of the fingers of both hands. This trouble had first been noticed about twelve months before, during the winter. As the warmer weather came on she became more comfortable, but as soon as the present winter started her fingers became cold, blue, tingling, and numb whenever the hands were exposed even for short periods to cold. Latterly the fingers had started 'going dead', and she began to find her work as a hairdresser increasingly difficult, so much so that she feared she would have to give it up. There were no past illnesses.

The girl was somewhat pale and fat, with a small goitre and sluggish peripheral circulation. The fingers and hands were cold, blue, and moist, but until she held them in cold water the fingers did not go 'dead', and after recovering they became congested and throbbing. The pulses were full and soft. Her feet were also cold, but did not cause any discomfort, and exhibited no vascular spasms or trophic changes. General examination otherwise revealed nothing abnormal. There was no radiographic evidence of cervical rib. A diagnosis of Raynaud's *disease* was made.

The patient failed to respond to various forms of physiotherapy, and it was decided to perform a bilateral cervico-dorsal sympathetic ganglionectomy. Unfortunately the pleura was torn on the second side, and she developed pneumonia. However, she recovered from this and the hands and fingers have remained warm and dry ever since. She is doing her work as a hairdresser, which entails using cold water sometimes, but this gives no trouble. She has a Horner's syndrome on both sides, as always happens when the inferior cervical ganglion is completely removed.

PATHOLOGICAL REPORT.—Professor Haswell Wilson says of the section of the tissue removed : " This shows diffuse fibrosis and degenerative changes in the ganglion cells."

Case 2.—V.B., age 27, male. Copper-smith.

Admitted to hospital in August, 1930, complaining of cramp-like pain in the right calf. This began two years ago, within a fortnight of appendicectomy. At

first he thought it was weakness from the enforced lying in bed, but even when he had otherwise returned to good health and had restarted work this pain came on about 5 o'clock every afternoon. It became gradually worse and started earlier in the day, and at the present time it begins as soon as he is out of bed, and after walking only a quarter of a mile he has to stop to rest. He gave up his work several weeks ago. The pain is of a 'crampy' nature' and he says the foot sometimes feels numb. There is never any pain in the left leg.

The patient is a strong healthy man with no history of any serious illnesses apart from appendicitis two years ago. He is not a heavy smoker. There is no muscular wasting, but the right foot feels colder than the left, it is somewhat dusky in colour, and the dorsalis pedis and posterior tibial pulses are only about half the volumes of those on the left side. General examination does not reveal any other abnormality.

A diagnosis of thrombo-angiitis obliterans was made, and, conservative treatment having failed, it was decided to perform a lumbar sympathetic ganglionectomy on the right side. This was done transperitoneally. The man's progress was rapid and he claimed to feel more comfortable in the right calf. For the first week or so after getting up from bed he experienced a slight cramp, but this soon disappeared and he returned to work.

On examining him in April, 1932, he stated that he was back at full work and never under any circumstances suffered any pain whatsoever. Both legs appeared exactly similar, though the right one is the warmer, and the pulses on the right side were as full as those on the left. He says he never perspires in the right leg, and this is reflected in the condition of the insides of the shoes, the right one being dry and shiny.

PATHOLOGICAL REPORT.—The report on the section of the tissue removed, made by Dr. F. W. Mason Lamb, states that, " The ganglion cells show degeneration. Many have lost their nuclei. There are small areas of hæmorrhage in the connective tissue ".

Case 3.—J. P., age 30, male. Turner.

. Admitted to hospital in August, 1930, complaining of extreme tenderness of the amputation stump of his right thumb. The thumb was crushed in a lathe in June, 1929. It was trimmed in the casualty department, but because of sepsis an amputation was performed. This became painful because of an adherent scar, and a second amputation was done. The stump was still tender and painful and the removal of amputation neuromata in no way improved his condition. I saw him for the first time in May, 1930. The stump was then so sensitive that if he caught it against his clothing he gave a shudder of pain : he was completely incapacitated.

On examination it was seen that the stump was blue, cold, clammy, and the skin was shiny and atrophic. This condition also involved the ball of the thumb. It appeared to be a true case of causalgia with very definite evidence of sympathetic over-action. In view of the very localized distribution it was decided to give a course of diathermy and massage, but there was no improvement after two months of this treatment. A cervico-dorsal sympathetic ganglionectomy on the right side was advised, and this was carried out ; the effect was immediate. The stump at once became warm, pink, and dry, and all tenderness and pain disappeared, and within a fortnight the skin had lost its atrophic appearance.

On examination in April, 1932, this healthy and comfortable condition was maintained.

PATHOLOGICAL REPORT.—The report by Dr. F. W. Mason Lamb states, " The ganglion cells vary very much in size. Some are shrunken and have lost their nuclei. The connective tissue appears to be increased."

Case 4.—S. O., age 41. Housewife.

Admitted in June, 1931, complaining of coldness of both hands, particularly of the right one, and stiffness of the fingers with consequent inability to do such things as sewing and knitting. The coldness commenced about ten years ago, but

was then diagnosed as chilblains; no beneficial treatment was found. There is often a burning throbbing pain in the hands, and when they are warmed they become cyanosed and ache. The condition becomes aggravated in cold weather. The fingers cannot be extended.

On examination both hands are found to be cold and a little moist. The thumb and all fingers on the right side are flexed and cannot be extended voluntarily, though passively they can, i.e., there is no contracture. The movements of the left hand are clumsy, but extension is not limited. There is diminution of all forms of sensation in the right fingers and hand as high as the wrist. Although the patient was kept in bed for the first few days after admission to hospital the hands were at no time observed to become really warm; they became cyanosed when warmth was applied. The radial pulses are small in volume. She is quite unable to perform any of the more complicated movements with the fingers of the right hand. The feet are also always cold but do not cause any serious discomfort.

On injecting 50 million T.A.B. intravenously the whole of the limbs became warm and pink within two hours, and remained so for several hours, and the patient voluntarily declared that she had not had such comfort for many years.

It was decided to remove the stellate and second dorsal sympathetic ganglia with their connections on the right side. There was uninterrupted recovery and the hand remained warm, dry, and pink, and was much more comfortable.

As seen in April, 1932, the comparison of the two hands is very remarkable, for whereas the left hand is still cold, blue, and stiff, the right is of normal colour, it only becomes cool when exposed to cold but never aches or feels stiff, and when warmed in water or by the fire it never 'goes black' like its fellow on the opposite side. Moreover, she can now extend the thumb and index and middle fingers and is able to sew and do many things which she had not done for ten years.

PATHOLOGICAL REPORT.—The histological report says, " There is some fibrosis of the substance with atrophy of a few of the ganglion cells." (G. H. W.)

This case was thought to be an example of Raynaud's disease, but atypical, in that the spasm appeared absolutely constant. The response to injection of T.A.B. seemed to confirm this, for the pulses became soft and full. An X-ray picture failed to reveal any evidence of a cervical rib, which diagnosis had been considered, it being thought that the patient resembled the cases recently reported by Telford and Stopford. The inability to extend the fingers and the sensory changes were thought to be due to the prolonged vascular change. However, now that the spasm has been released by the operation it is realized that there is an ulnar paralysis present; and the diagnosis is almost certainly one of cervical fibrous band occurring in a patient whose sympathetic nerve-supply to the brachial plexus runs in the lowest trunk of the plexus, as described by Telford and Stopford. She has been advised to have a second operation for this, and has received such benefit from the first operation that she has accepted.

Case 5.—E. B., age 27. female. At home.

Admitted to hospital in early October, 1931, complaining of backache, difficulty in walking, pain and numbness of the hands and feet, and trophic ulcers of the feet, over a period of seven years; excessive perspiration of the legs during the last eighteen months.

The backache and part of the difficulty in walking were due to bilateral congenital dislocation of the hips and bilateral pes cavus, for which conditions she had been operated on at various times by Mr. Naughton Dunn with satisfactory results. During the same time the circulation of the legs in particular, but of the hands, too, became progressively worse, so that the hands and legs were always cold, blue, and numb, and from time to time shallow painful ulcers appeared in the region of the heels and ankles. These always healed with rest in bed, but tended to break down as soon as the patient attempted to get about again. The legs ached a great deal. During the past eighteen months the perspiration in the legs became so copious that her bedclothes were damp each morning. The hands also perspired a great deal, and the fingers tingled and were always cold and blue. These vasospasms never seemed to relax.

On examination the legs are mottled with various shades of blue and red : the capillary circulation is sluggish ; the skin is cold, feeling little above room temperature ; there is an ulcer 2 in. in diameter on the left heel and another of ½ in. in diameter over the right external malleolus : these ulcers are very tender on the slightest pressure. The bedclothes around the feet are damp from perspiration, which can be seen trickling off the feet and lower halves of the legs. These vascular changes seem to cease above the knees, though the whole body and upper limbs are moist with perspiration. The legs below the knees show an impairment of all forms of sensation and there are patches of complete loss of tactile sensation. The dorsalis pedis arteries cannot be felt, and the posterior tibial arteries give very thin pulses. The vascular spasm has not been observed to relax at all.

Fifty million of T.A.B. intravenously caused a rise of temperature in the mouth from 98° to 100·4° in two hours, and in the feet from 68° to 96° in six hours—a rise of 28°. It was decided to advise a bilateral lumbar sympathectomy.

This was carried out and the patient made an uninterrupted recovery. From the moment she left the table the lower limbs were warm, dry, and pink, and they have remained so ever since. The ulcers had both healed within a week's time. She was allowed up at the end of a fortnight and then walked quite well. She declares that the feet have been more comfortable since operation than for many years, and now asks that something similar may be done for her cold hands.

PATHOLOGICAL REPORT.—The report states, "There is distinct inflammatory cell infiltration and many of the nerve-cells have lost their nuclei and staining reactions." (F. W. M. L.)

Case 6.—N. H., age 22, female. Housework.

I had the opportunity of operating on this patient in collaboration with my senior colleague, Mr. J. B. Leather, under whose care she was admitted.

Admitted to hospital in March, 1931, with the complaint of obstinate constipation over a period of more than seven years. She had had a long history of surgical and medical treatment, of which the following are the main points. Appendicectomy in 1923 for chronic appendicitis. Following this she began to be troubled by constipation, but managed to get regular movements with various aperients. Six months later the tonsils and adenoids were removed. In 1924 she had an illness which she describes as 'ulcerated bladder' and was in hospital for eleven weeks. After this she developed 'stomach trouble' and the constipation became more troublesome. She was taken into another hospital in 1928 where a thorough X-ray examination of the abdomen was carried out but nothing abnormal was found. She was then examined periodically in the out-patient department, and various forms of treatment were advised for the constipation, which was getting worse and which had reached the stage of requiring enemas on alternate days ; if these were omitted, she would go for ten days without evacuating the bowel and would then be forced to have recourse to enemas in order to get comfortable again. She was once again admitted to the wards, but no treatment was of any use, and it was decided to resect the presacral and lumbar colonic nerves on the assumption that this was some form of achalasia.

The operation presented no difficulties and the lumbar colonic nerve was found as a sturdy single branch running along with the inferior mesenteric artery. A slender adhesion between the cæcum and middle of the transverse colon was cut and tied off.

The patient began to have easy bowel movements from within a few days of operation. When interviewed in April, 1932, she stated that this satisfactory state of affairs had been maintained, and this without the aid of enemas or laxatives.

It may be argued quite reasonably that this is not a fair case upon which to pass judgement, as a definite adhesion was severed. This adhesion was very slender and about 3 in. long, and it certainly did not cause any kinking of the colon or cæcum. Moreover, since an enema would always produce a good result before operation it can be safely assumed that the contents of the colon passed the area of adhesion without difficulty, and were in fact held up in the pelvis.

PATHOLOGICAL REPORT.—" No abnormality is seen in the ganglion cells, but a collection of lymphocytes is seen in the neighbouring tissues and there is some evidence of chronic inflammation of the connective tissue in the region of the nerve." (F. W. M. L.)

Another case of causalgia, almost exactly the same as *Case 3*, in history, signs and site, was treated by a dorso-cervical sympathectomy. For three weeks the result was perfectly satisfactory, the stump becoming warm, dry, pink, and painless, but the patient then relapsed and evidence of vascular spasm accompanied the return of extreme tenderness. I believe the anatomical basis for this operation to be so sound that I attribute this failure not to faulty theory but to incomplete execution. This particular operation was performed through the second transverse process and rib, and the inferior cervical ganglion was only partially removed ; the fact that the patient did not show a Horner's syndrome supports this view. It is hoped that he will submit to a second operation.

I was also persuaded to try this type of treatment for a case of severe intermittent claudication in one leg. The man, age 56, had arteriosclerosis, but there were no vascular spasms observable. He recovered perfectly from the operation, but there was not the slightest improvement as regards pain. This was an early case and the fever test was not applied. Such a result therefore should not be held to condemn the operation ; it was an ill-chosen case and incompletely investigated.

I have, more recently, performed one or other of these operations in a number of other cases, and though the results promise well it is as yet too early to claim them as examples of permanent relief.

COMMENTARY.

It is realized that the method of recording surface temperatures by bandaging the bulb of a laboratory thermometer between adjacent fingers or toes is a rough one as compared with those of the electrothermal couple or the calorimeter, but for practical purposes it is sufficiently accurate ; for experimental work it most certainly is not.

It is a matter of no small interest that the thickness of the sympathetic cords intervening between ganglia varies in different patients; sometimes they are as thick as the ulnar nerve in the arm ; at other times they are no bigger than the posterior interosseous nerve. In some cases there seems to be evidence of old inflammation around them in the form of adhesions, and in *Case 5* there were signs of recent inflammation in the form of round-celled infiltration. The common finding of changes within the ganglia themselves is of great interest too, and suggests that they may have been the seat of some infective or toxic process. Investigations into these problems are being pursued.

SUMMARY.

1. If it is assumed, as it is by many authorities, that vasospastic conditions are due to overaction of the sympathetic nervous system, then the operation of peri-arterial sympathectomy as an attempt to denervate the whole arterial tree of a limb is anatomically unsound. Nevertheless, Leriche's

operation has its uses in cases of temporary interference with the nutrition of limbs, and may also prove beneficial in those patients requiring full sympathectomy but who are not in a fit state for the major procedures.

2. Complete sympathetic denervation of the upper limb demands removal of the inferior cervical and 1st and 2nd dorsal ganglia together with the intervening chain : that of the lower limb requires removal of the 2nd, 3rd, and 4th lumbar ganglia plus the intervening chain.

3. The sympathetic control to the sigmoid colon can be abolished by excision of the presacral nerve, or by severing the lumbar-colonic nerve.

4. Before deciding upon sympathectomy in vasospastic conditions it must be quite certain that the case presents irrefutable evidence of sympathetic hypertonicity—that is, an upset of the normal sympathetic-parasympathetic balance. This can be proved by inducing a fever in the patient and noting the degree of vasodilatation which ensues.

5. Cases illustrating the employment of this form of treatment are recorded.

6. Comment is made upon the appearance of pathological changes within the ganglia removed.

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ACUTE PERFORATED PEPTIC ULCER.

A REVIEW OF 64 CASES.

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THIS study was undertaken with a view to finding out, in particular, the late results of acute perforations of peptic ulcers. Incidentally other data were given notice and are recorded below. It is often stated that acute perforation of a gastric or duodenal ulcer, given operation, leads to its complete healing. If this is the case, both doctors and afflicted patients should regard the onset of perforation as a blessing in disguise—provided, of course, that the patient is given a reasonable chance of recovery by early operative treatment. Such a state of affairs seems at best too good to be true, while at least it warrants investigation.

The number of cases investigated was 64. All were operated upon by one of us (J. G.) during the years 1922 to early 1929, so that none has a post-operative history of less than two years, and in 24 cases the period is five years or over.

Sex Incidence.—Of these cases, 58 were males and 6 were females, a proportion, roughly, of 10 to 1. These figures bear out a fact which we are sure is the experience of every surgeon who has dealt with much much emergency surgery—namely, the preponderance of males over females amongst the individuals suffering from this acute abdominal catastrophe. For example, Urrutria[58] records 49 cases in males out of a total of 52, Dineen[13] 138 males in 142 cases, Hinton[27] 103 males in 105 cases, and Dunbar[16] 355 males in 387 cases. Other authors who have recorded a series of cases, are Brown,[5] Kreuter,[30] Platou,[45] Robitshek,[47] and Williams and Walsh,[60] all of whom find sex relativities similar to those recorded above.

Localization.—*Table I* shows the site of the ulcer in the cases of the present series.

Table I.—LOCALIZATION IN 64 CASES OF PEPTIC ULCER.

	DUODENAL	PYLORIC	GASTRIC
Males	52	4	2
Females.. ..	6	—	—
Totals ..	58 (90·7%)	4 (6·2%)	2 (3·1%)

While in most cases of acute perforation the exact site of the ulcer can be recognized with relation to the pylorus, in some the scarring and induration have obliterated the pyloric vein of Mayo or are masking the definition of the thickened muscle constituting the pyloric sphincter. thus making it impossible to identify the site definitely as duodenal or gastric. These latter cases are inserted into the table above as 'pyloric'. In other cases. of course. the ulcer is pyloric. not by a process of elimination but in point of fact.

The figures in the table are worthy of note. What strikes us most is the large percentage of duodenal as opposed to pyloric or gastric ulcers. We would remark here that the number of perforated ulcers operated upon at the Royal Victoria Infirmary. Newcastle-upon-Tyne. is very large (in 1928. for example. the number was 210 : in 1929. 221). and when glancing through any volume of notes one cannot fail to notice the large percentage (usually in the neighbourhood of 90 per cent) of duodenal ulcers in cases of acute perforation. The percentage in this series is 90·7. In view of this it is interesting to note in the literature articles on the subject recording a much smaller percentage of duodenal ulcers. Lewisohn.[32] for example. reports only 18 ' duodenal and pyloric ' ulcers in 33 cases (55 per cent) ; Bryce[7] 100 duodenal ulcers in 153 cases (65 per cent) ; Deaver and Pfeiffer[11] 38 duodenal ulcers in 55 cases (69 per cent) ; while Gibson.[19] Judine.[29] and Fermand[18] record the low percentages of 55, 48, and 40 respectively in their series. McCreery[34] reports a preponderance of gastric ulcers over duodenal ulcer in his cases. 15 being gastric and 10 duodenal.

In all our duodenal cases. the ulcer was situated on the anterior or antero-superior surface of the duodenum. Both gastric ulcers were situated on the lesser curvature near the cardiac end. There was no case of multiple perforations, of multiple duodenal or gastric ulcers, or of associated duodenal and gastric ulcers.

While it is the commonly expressed opinion that in cases of acute perforation in females the ulcer is more often gastric than doudenal, in the series we are reporting all six females had perforated duodenal ulcers, while the only two cases of perforated gastric ulcer occurred in males.

Age Incidence.—*Table II* gives the age incidence in our series of cases.

Table II.—AGE INCIDENCE IN 64 CASES OF PEPTIC ULCER.

AGE	MALES	FEMALES	TOTALS
10–19	2	—	2
20–29	11	2	13
30–39	18	1	19
40–49	14	2	16
50–59	9	—	9
60–69	4	1	5
Total	58	6	64

The oldest male was 67, the oldest female 69 ; the youngest male was 17, the youngest female 28. The average age of the males was 40 years ; of the females 41·5 years.

In more detail it may be stated that both cases of gastric ulcer are included in *Table II*; they were aged 25 and 55 respectively, giving an average age of 40 years. Therefore, had they been considered separately, the average age of the duodenopyloric group would still be 40.

From these figures it is seen that the most prevalent decade of life in which the perforation occurred in this series is the fourth, while the third, fifth, and sixth decades are strongly represented. The number of cases occurring in the more extreme decades is almost negligible, but it proves that acute perforation of a peptic ulcer may occur at almost any age, in regard to which Smith[54] makes the interesting statement that acute perforation has been reported in an infant of 2 months and in a woman aged 77. Harrison[24] recently reported a case of perforated duodenal ulcer in an infant 10 weeks old and gives references to eight authors who have each reported a case in infants under one year. The further bearing of this factor on the remote results of acute perforated ulcer will be considered later.

Brown,[5] Fermaud,[18] and Robitshek[47] each shows in his series an age incidence which runs parallel to that recorded by us.

Length of History.—A consideration of the length of history in these cases is of interest, as it shows that it can be divided into two distinct groups. In 56 cases (88 per cent) a history was obtained of gastric symptoms—usually the typical ulcer syndrome—extending over a period of years, while in the much smaller group of the remaining 8 cases, 4 had a history of stomach trouble for only three or four weeks, and in the other 4 the acute pain of perforation was the initial symptom.

Amongst others who have made note of the presence of a long history in the great majority of cases of acute perforated ulcer are Colp,[9] Henry,[25] Moynihan,[37] Sherren,[52] Dineen,[13] and Robitshek.[47] It seems obvious, then, that the length of history has a definite bearing upon the perforation incidence of peptic ulcer, and one cannot but come to the conclusion that the older the ulcer becomes, the more likely it is to perforate.

Treatment.—In this series all the cases were treated by simple suture, the ulcer thereby being infolded, except in one case in which gastro-enterostomy was performed in addition to the suture of the ulcer.

The closure of the perforation was brought about by the following standard procedure. Several interrupted Lembert catgut sutures (the number depending upon the size of the ulcer, but on the average 3 to 5) are passed over the ulcer from side to side parallel to the long axis of the duodenum, bites being taken in healthy tissue as close to the sides of the ulcer as possible. When these are tied, the ulcer is infolded and the perforation closed, the line of closure being at right angles to the longitudinal axis of the abdomen. As an extra precaution against leakage, a continuous Lembert suture of catgut is superimposed to invaginate the line of closure. Small tags of omentum, if readily available, may be brought into use by being stitched over the suture line—this is merely a refinement and not an essential in the closure of the perforation. We do not claim that this type of suture has any particular advantage over the ordinary purse-string (unless it be that it interferes less with the blood-supply to the edges of the sutured ulcer), and there is no doubt that the type of suture used is largely a personal matter. The danger of

immediate stenosis following the infolding of the ulcer by either method is apparently negligible.

After the ulcer has been sutured, the peritoneal cavity is then mopped out and the abdominal wound closed. We do not consider drainage necessary unless there is a large amount of fluid present with much soiling and infection of the viscera, when a suprapubic drain is inserted for twenty-four to forty-eight hours.

Results.—

1. *Immediate Results.—Table III* shows the mortality in the present series of cases.

*Table III.—*IMMEDIATE MORTALITY IN 64 CASES OF PEPTIC ULCER.

TIME AFTER OPERATION	MALES	DEATHS	FEMALES	DEATHS
Under 12 hours	47	1	4	0
Over 12 hours	11	2	2	0
Totals.—				
Under 12 hours	51 cases with 1 death (0·5 per cent)			
Over 12 hours	13 ,, ,, 2 ,, (15·0 per cent)			
Grand Total.—	64 ,, ,, 3 ,, (4·7 per cent)			

All three deaths were in cases where the ulcer was duodenal, although the gastric cases, generally considered to be more lethal, were operated on ten and fourteen and a half hours respectively after perforation. It will be seen from the table that the mortality affected only the males.

The results shown corroborate a commonly accepted fact—namely, that the longer the time between the moment of perforation and operation, the higher will be the resulting mortality.

2. *Remote Results.—*Of the 61 cases remaining out of the 64 operated upon, we were unable to trace 13 (including both cases of gastric ulcer), leaving 48 of whom we have knowledge. Four have died since the operation, the causes being respectively cardiovascular disease, bronchopneumonia, recurrent perforation with fatal peritonitis five months after the previous operation, and sudden death from an unascertainable cause about five weeks after operation, the patient being up and about and feeling quite well to within a few minutes before his death.

The results of the remaining 44 cases are as follows :—

Satisfactory ..	..	17	38·6 per cent	}	63·6 per cent
Fair..	..	11	25·0 ,, ,,	}	
Poor..	..	16	36·4 ,, ,,		

All these cases were treated by suture alone. The one case treated by suture and primary gastro-enterostomy is unfortunately numbered amongst those whom we have been unable to trace.

A note of explanation with regard to the terms 'satisfactory', 'fair', and 'poor' is necessary :—

By 'satisfactory' is meant symptomatic cure. The word 'cure' in describing the results of these cases is purposely avoided, for one cannot assume that the absence of symptoms means necessarily that the ulcer has healed, for we must acknowledge the existence of the silent ulcer.

By 'fair' is meant return of symptoms in a degree not sufficiently severe

to prevent the patient's going about his work and being a useful member of the community. Many of these patients are so little troubled that they refuse to submit to a further operation.

What is the underlying pathology in these cases? Symptomatology can help us only a little. For instance, 2 of these cases have had a mild attack of hæmatemesis, while 2 others have noticed black stools on one occasion. We think we can reasonably assume that in these cases the ulcer was still active at the time when these symptoms were noted. In 4 cases slight pain only at irregular intervals was complained of. The pathological significance of this is doubtful, for it by no means necessarily indicates the presence of an active ulcer. The same may be said of the occasional vomiting complained of by 3 other cases. A barium meal might help to elucidate the pathology, but in this respect it must be remembered that a deformity of the duodenal bulb can be caused by a healed ulcer as well as one which is active.

By 'poor' is meant a return of symptoms severe enough to incapacitate the patient. In some cases the symptoms are those referable to an active ulcer, in others they are indicative of the onset of the complication of pyloric stenosis.

Interpreting the results, we see that in this series a little over a third have had no further trouble at all, a quarter can get along well enough to refuse further operation, and a little over a third have received no lasting benefit at all from operation.

In the paragraph on the 'length of history' we made mention of 8 cases, 4 of whom had a history of stomach trouble for only three or four weeks, while in the other 4 the acute pain of perforation was the initial symptom, and stated that the bearing of this factor on the end-results would be considered later. It was of great interest to us to be able to trace 6 of these cases and to find that in all the result is satisfactory.

Perhaps even more interesting is the fact that in 7 of these 8 cases the pathology found was that of a chronic ulcer—that is, induration around the perforation—proving that an ulcer may be present some time before it gives rise to either symptoms or complications.

Secondary Operations.—Judging by the literature, the majority of surgeons find that after simple suture a certain percentage of cases develop symptoms suggestive either of a recurrent ulcer or of complications, and of sufficient severity to necessitate further operation. The percentage of such cases in the present series is 36·4—a little over a third. This figure agrees with that of Pool and Dineen.[46] Brenner,[4] Smith,[54] and Southam[62] have each reported a smaller percentage of these cases. Stewart and Barber[56] are exceptional in that they have not found it necessary to perform a single secondary operation.

On the 16 cases (36·4 per cent) whose results have been classified as poor, 10 have had a second operation performed. In 8 of the 10 gastro-enterostomy was done and the results are as follows :—

Satisfactory	..	..	3	(37·5 per cent)
Fair	..	..	2	(25·0 per cent)
Poor	..	..	3	(37·5 per cent)

The results run parallel with those obtained after simple suture—a little

over a third have had no further trouble, a quarter of the number improved, and a little over a third have received no benefit.

Recurrence of Symptoms after Operation.—*Table IV* shows the times of recurrence of symptoms in the 27 cases comprising the fair and poor results.

Table IV.—Post-operative Recurrence of Symptoms in Peptic Ulcer.

Months after Operation	Number
0–6	7
7–12	5
13–18	6
19–24	3
Over two years	6

These figures show that in over 50 per cent of these cases the onset of the recurrence of symptoms did not take place until over a year had elapsed after their operation, and in nearly 25 per cent the onset was delayed to over two years—in 2 cases three and a half and five years respectively. Hence we must disagree with Bryce,[7] who holds the view that any return of symptoms after simple closure of a perforation will take place within two years. Furthermore, the figures in the above table in themselves sound a warning note in regard to the too early publication of results claiming 'cures' after treatment of perforated ulcer.

Review of the Surgical Procedures Adopted in Cases of Acute Perforated Ulcer.—Perhaps it is not out of place to discuss the various types of operative procedures which are used in the treatment of acute perforated ulcer and to give a brief historical outline of their development. They may be summarized under four headings: (1) Suture alone; (2) Suture followed by (primary) gastro-enterostomy; (3) Excision of the ulcer and suture (pyloroplasty); (4) Partial gastroduodenal resection.

We have read that Roux[48] in 1883 performed the first operation for perforated ulcer, that Mikulicz[35] is given the credit for the first (unsuccessful) suture of a perforated ulcer in 1889, and that Kriege,[31] in 1892, was the first to suture such an ulcer with success. For several years this simple procedure remained unassailed as the standard method of treatment, until Braun[3] in 1897 introduced the performance of gastro-enterostomy in addition to suture of the ulcer. Moynihan[36] in 1901 urged that a gastro-enterostomy should be performed at the time of closure in order to counteract the pyloric stenosis which sometimes followed the operation of infolding of the ulcer by suture. Deaver[10] has proved himself the great champion of this operation in the U.S.A. Ever since its inception, the performance of this further additional surgical procedure has been, and still is, the subject of a great deal of controversy.

It is of interest that Guthrie,[22] who sent out a questionnaire to numerous American Surgeons with regard to the advisability of performing primary gastro-enterostomy, says it is obvious that by far the greater number are conservative and employ simple suture only.

Suture of the ulcer alone or combined with a primary gastro-enterostomy are both only palliative measures, and soon the idea of a radical treatment bore fruit. It was introduced first in 1909 by Dowden,[15] who excised the ulcer in the longitudinal axis of the duodenum and sutured the opening, thus formed, in the transverse axis. This operation had the double advantage of not only removing the ulcer but also of actually widening the lumen of a hollow viscus at a point where stenosis reasonably might have been expected.

Bolder spirits in time conceived the idea of extending the area to be removed so as to include the whole of the ulcer-bearing area of the stomach and duodenum. Haberer,[23] in 1919, was apparently the first to attempt to popularize resection in cases of perforated ulcers, and was followed soon afterwards by Eunike,[17] Massari,[33] and Schwarzmann,[51] who all reported further successful cases, so that partial gastrectomy and duodenectomy as the treatment for acute perforated ulcer is of quite recent development. Its practice has been confined, so far as we can ascertain, almost entirely to Continental clinics, and is not indulged in to any extent by British or American surgeons. Not every Continental surgeon, however, is in favour of resection, for both Brunner[6] and Noetzel[40] condemn the procedure as unnecessary and dangerous.

Each of the four methods of treatment has its advocates and its opponents, and since this is so, it is obvious that there is still a wide divergence of opinion on the subject of the treatment of acute perforated peptic ulcer. Perhaps it would be well, at this point, to examine the claims put forward by the advocates of each method in proof of its superiority over the others.

Everyone realizes that the acute perforation of a peptic ulcer is a catastrophe of such magnitude as to threaten seriously the life of the afflicted patient. The hypothesis that it is sufficient to ward off the immediate danger without taking thought of the future forms the main consideration upon which is based the claim that *simple suture* of the ulcer is the treatment indicated. The patient in his precarious state is thereby subjected to the least possible amount of operative interference, and the prognosis, so far as life is concerned, is improved. In other words, any operative procedure other than suture is not only unnecessary, but, by prolonging the operation and tending to spread infection in the peritoneal cavity, endangers the life of the patient. Further, immediate stenosis following the suture of an ulcer is, to all intents and purposes, unknown to occur, and cases in which it develops at a later date can then be dealt with accordingly. In this respect, one surgeon has remarked that it is better to have a living patient with pyloric stenosis than a dead one without. Hæmorrhage and recurrence of perforation are also spoken of as being exceptional sequelæ after simple suture. It has been claimed that a perforated ulcer, having been sutured, then undergoes cure in nearly all cases —one of the strongest arguments—if it were true—in favour of suture as the best treatment.

There arises next the advisability of performing a (primary) *gastro-enterostomy* at the time of suture. The arguments against this—as against *any* procedure further than suture—have been indicated above. However, those who are in favour of this form of treatment point out that, in the first.

place, it does not add to the mortality in early cases in spite of the prolongation of the operation and of the accusation that infection will be spread, but may actually be instrumental in reducing it. Secondly, they argue that even if no immediate stenosis develops after simple suture, at all events gastro-enterostomy relieves the tension on the suture infolding the ulcer and at a later date guards against hæmorrhage, recurrence of perforation, and the effects of a subsequent stenosis which may occur. Thirdly, they say that the anastomosis is beneficial by permitting of increased alkalinization of the gastric juice. Fourthly, that suture alone does *not* cure the ulcer in the high percentage of cases claimed by its advocates. Fifthly, that the incidence of gastrojejunal ulcer, the bugbear of gastro-enterostomy, is very low.

The reasons given by Dowden[15] for his introduction of *excision* of the perforated ulcer were twofold. In the first place, the ulcer is got rid of entirely or at least to a great extent, and, secondly, the healthier tissue exposed is much more suitable for rapid and safe suturing. Moreover, by performing a plastic operation when suturing up the opening made by the excision of the ulcer, the lumen of the gut is actually increased in a place where following simple suture a stenosis might reasonably be expected to occur at a later date. The disadvantages are obvious and are mentioned by Dowden himself. Only specially selected cases can be dealt with in this manner, others are too unapproachable and at the best difficult to close; any fixity of the ulcer by adhesions to the posterior abdominal wall also tends to make the operation very lengthy and difficult, if not impossible.

The main reasons for the adoption of *partial gastrectomy* as the treatment for perforated ulcer are : first, the success which has attended the treatment of chronic ulcers by this method ; secondly, dissatisfaction with the results obtained by the palliative or less radical procedures of suture alone, suture with primary gastro-enterostomy, and local excision followed by pyloroplasty ; thirdly, that removal of the ulcer-bearing area definitely saves the patient from such complications as hæmorrhage, recurrence of perforation, or pyloric stenosis. The performance of such a lengthy and severe operation on a patient acutely ill from such a condition as a perforated ulcer, when a much simpler one is all that is necessary to save the patient's life, is strongly opposed by many surgeons who uphold the view that the first principle of emergency surgery is to ward off the immediate danger, without considering the future.

Comparison of Results—Early and Late.—On the whole the mortality percentage of the present series is eminently satisfactory, especially when compared with that of other series of cases. Statistics, of course, are unsatisfactory, for, after all, each case is an individual with a susceptibility and power of resistance to disease peculiar to himself and not admitting of comparison with that of his fellow-sufferer. However, they are the only means we possess in our endeavours to assess the value of any particular operative treatment.

In an article of this length we can give the mortality percentages of only a limited number of series of cases after treatment by the different methods. In considering figures representing total mortality, series with a larger number of late cases will naturally have higher percentages—another factor which tends to decrease in value statistics appertaining to the condition under consideration.

IMMEDIATE RESULTS OBTAINED BY OTHER AUTHORS AFTER THE VARIOUS METHODS OF TREATMENT.—

1. *Simple Suture.*—The following list shows the total mortality percentage in series of cases treated by simple suture of the ulcer, and are taken from recent articles on the subject: Brown[5] 39, Deaver and Pfeiffer[11] 17, Dunbar[16] 29, Gibson[19] 18·6, Morrison[38] 47, Moynihan[37] (duodenal cases) 38, Platou[45] 20, Smith[54] 12, Southam[62] 9, Stenbuck[55] 38·3, Stewart and Barber[56] 8·3, Turner[57] 14·6, Urrutria[58] 15·7.

From this it would appear that an average mortality following this method of treatment is 20 to 30 per cent. Amongst these authors the few who published tables showing the relationship of mortality to the time of operation after perforation all prove by their figures that the earlier the operative treatment is undertaken the less the mortality. For instance, although Southam's total mortality was 9 per cent, 34 of his cases operated on under twenty-four hours all recovered.

2. *Suture and Primary Gastro-enterostomy.*—The figures representing mortality percentages in cases treated by suture and primary gastro-enterostomy present some interesting contrasts to those shown in the last paragraph. Brown,[5] Deaver and Pfeiffer,[11] Morrison,[38] Moynihan,[37] Platou,[45] and Stenbuck[55] all report a smaller percentage of mortalities, this being most marked in the cases of Deaver and Pfeiffer (5·5 per cent by this method compared with 17 per cent of the suture alone), and Morrison (3 per cent—47 per cent after suture alone), while Platou, whose mortality after simple suture was 20 per cent, had no deaths by this method. On the other hand, Urrutria showed an increase in his mortality-rate by over 4 per cent, and Judine[29] and Walton[59] had respective rates of 38·8 and 35·2 per cent.

3. *Excision of Ulcer and Pyloroplasty.*—The reported series of cases treated by excision and pyloroplasty consist of small numbers only, the largest being that of Moynihan,[37] these being 22 cases with 1 death, a mortality of 4·5 per cent. Dowden[15] and Grimault[21] had no deaths. Hinton[26] had a mortality of 8·3 per cent, while that of Williams and Walsh[60] was no less than 33 per cent. Bager,[1] of Stockholm, did 78 cases with a mortality of 11·5 per cent, and lost none of 45 cases operated on within six hours after perforation.

4. *Partial Duodenectomy and Partial Gastrectomy.*—The results shown by men from Continental clinics who prefer radical measures in the treatment of acute perforated ulcer demand the closest consideration, especially in view of the fact that at the present time the great majority of surgeons show a distinct preference for conservative surgery and are inclined to condemn resection unreservedly.

Judine,[29] of Moscow, tells us that he has sought to do resection in all cases except those in which: (1) Intoxication is advanced; (2) The patients are too old; and (3) Resection is difficult. As a result of his activities he has performed 47 resections with 6 deaths, a mortality of only 12·7 per cent; moreover, in 2 of these cases total, not partial, gastrectomy was done, and both patients survived. He attributes his success largely to the use of splanchnic anæsthesia. Pierre Duval, in his discussion of Judine's results,

comments favourably on 'this magnificent series', considering it an unanswerable argument in favour of this resection treatment for perforated ulcer which he had tried to establish in a thesis three years previously.

Doberauer,[14] of Carlsbad, records 11 resections for perforated ulcer with 3 deaths (27 per cent mortality). Odelberg,[41] of Stockholm, has performed 20 partial gastrectomies for perforated ulcer with only 1 death (5 per cent mortality). His only death occurred in a case which had been perforated seventeen hours, and he attributes the fatality to having seen the patient first when it was too late. Mulleder and Neuberger[39] reported 21 resections with 1 death (4·7 per cent mortality). Hromada and Newman[28] report 19 resections with 14 recoveries (26 per cent mortality). They state that all these cases showed widespread peritonitis, and in all more than two-thirds of the stomach was removed. Schwarz[50] reports a series of 10 cases : 9 of these were operated on under twelve hours after perforation and survived. His only death occurred in a patient who had been perforated for eighteen hours.

Every surgeon must be impressed by the figures quoted above, since in some series the mortality is less than that resulting from the treatment of the perforated ulcer by the simplest possible method—namely, laparotomy and suture. It is obvious, however, that such results can only be obtained where the operator has more than average ability and experience in gastric surgery, quite apart from the fact that the patient's general condition must be good.

The sphere of partial gastrectomy as the treatment for perforated ulcer must necessarily be always restricted to clinics specializing in gastric surgery. We are quite sure that it would be unwise for the average general surgeon to attempt to establish this large resection operation as his routine treatment for perforated ulcer, and think he would be well advised to content himself with some less formidable procedure. We think our experience of a 0·5 per cent mortality in cases operated upon under twelve hours, with a total mortality of 4·7 per cent, fully justifies the simplest operative measures—that is, suture alone. .

REMOTE RESULTS OBTAINED BY OTHER AUTHORS AFTER THE VARIOUS METHODS OF TREATMENT.—

1. *Simple Suture.*—Much has been made of the claim that suture of a perforated ulcer leads to its subsequent cure. Stewart and Barber,[56] for example, found that all of their 22 cases 'during the past five years' which had recovered from operation (suture alone) were well enough not to acknowledge that they were sufficiently uncomfortable to allow themselves to be re-operated on. Gibson[19] states that " In general, patients who recover from a perforation are cured of the ulcer. The great majority are restored to good health and are free from stomach symptoms " : and he adds that these patients do so well that he has been led to refrain from doing probably unnecessary operations as a routine. He followed up 93 patients, the results being 'excellent' in 41 and 'satisfactory' in 31.

In Southam's[62] series, 28 out of 37 patients have remained free from symptoms 'up to periods of two years' after simple suture, and 5 have slight symptoms only. Smith reports that in only 4 cases out of 35 which recovered from the operation was he asked for further advice some months after discharge. Pool and Dineen[46] report 60 per cent of patients well of those who

have recovered from operation (suture alone). Some of these had been operated on twelve years previous to publication.

Platou[45] reports results after suture not nearly so good as those above. Of 25 cases, 3 were re-operated on for perforation within a year, 8 have had gastro-enterostomy done since because of return of symptoms, and 8 are complaining enough to be constantly under medical supervision, so that only some 25 per cent showed anything like a satisfactory result. Pannett[43] believes that the true percentage of cures following simple suture is about 50.

Although we do not agree with those who state that simple suture of a perforated ulcer will lead to its ultimate cure in every, or nearly every, case, yet our personal experience leads us to believe that 60 to 70 per cent of patients treated in this way will have little or no further gastric symptoms. Hence we hold the view expressed by Pool and Dineen,[46] who consider that a definitely indicated later gastro-enterostomy is preferable to subjecting all cases to this procedure as a prophylactic measure, thus saving many patients unnecessary subjection to the possible dangers and discomforts of the operation.

2. *Suture and Primary Gastro-enterostomy.*—From our own series we cannot claim any familiarity with personal results following the performance of suture and primary gastro-enterostomy for perforated ulcer, but it is of interest to examine, for purposes of comparison, those of other writers.

Collinson[8] found that of 27 cases treated in this manner, 70 per cent are well, while of 26 cases treated by suture alone 65 per cent are well, most of the remainder having had gastro-enterostomy with satisfactory results. Deaver and Pfeiffer[11] report satisfactory results in all of 17 cases subjected to primary gastro-enterostomy, and are of the opinion that accumulated statistics and more accurate follow-up records are dispelling the fallacy that a successful operation for perforation cures ulcer, and tend to show that primary gastro-enterostomy lessens the likelihood of future ulcer symptoms and complications. Deaver,[10] we know, has long been the great American advocate of primary gastro-enterostomy in cases of perforated ulcer. In his latest writing he confirms his opinion, though for the past two years he has been making a wide removal of the pyloric sphincter muscle. Dineen[13] obtained 70 per cent good results with suture and primary gastro-enterostomy, as against 80 per cent good results following suture alone.

Lewisohn,[32] who has had experience of both methods, found that closure of an acute perforation, either with or without gastro-enterostomy, failed to cure in 39 per cent of cases. Fermaud[18] claims 90 per cent cures with suture and gastro-enterostomy and 50 per cent cures with suture alone. Platou[45] has done 15 cases in this manner with 100 per cent good results. Urrutria,[58] in 12 cases, has had to perform secondary operations for gastrojejunal ulcer in no fewer than 4—truly a high percentage. He also quotes more than twenty authors who have reported cases of gastrojejunal ulceration following gastro-enterostomy for acute perforating ulcer, and believes that the danger of secondary ulceration is a real one. Williams and Walsh[60] report that in both cases in which primary gastro-enterostomy was done there was a recurrence of perforation at the same site.

3. *Excision of Ulcer and Pyloroplasty.*—The operation of excision of the ulcer, when situated in the duodenopyloric region, followed by a plastic

operation. never seems to have gained much popularity since its introduction by Dowden.   It has, however, some staunch enthusiasts.

Dunbar[16] says that the operation has yielded splendid results and does not take more than two minutes longer than simple suture.   He also states that no case in which this operation was done has returned complaining of stomach symptoms. and that several of these cases have been X-rayed. when no delay has been shown in the stomach.

Grimault[21] has done 10 cases over a period of ten years. and all of them are well.   He says the operation is indicated in late cases where suture or infolding is difficult.   Although Williams and Walsh[60] reported a large mortality in cases treated by this method. yet in 5 out of their 6 surviving cases the result has been satisfactory.   Other authors who speak well of the operation. and show encouraging results. are Dehelly.[12] Girode,[20] and Basset.[2]   On the other hand. Hinton[26] has followed up 9 cases and found 22 per cent well.   In view of these results he is of the opinion that one is not justified in continuing to use this type of operation.   Rowlands and Turner[19] quote 12 cases treated by this method. of which 5 have had recurrence of symptoms.

4. *Partial Resections.*—Those who have published late results after partial gastrectomy are very enthusiastic about them.   Hromada and Newman[28] have followed up 13 of their 14 surviving cases for one to two years. and find that all are free from gastric distress. have good appetites. and have put on weight.   Furthermore, all have returned to their previous occupations. Their experience thus leads them to consider resection. even in the presence of peritonitis. the best treatment, and that older methods should be used only when no other method is available.   Paul.[44] from von Haberer's Clinic. considers that follow-up reports show this to be a more satisfactory operation than less radical measures.   Odelberg[11] has traced 8 of 10 cases who have been operated upon over a year.   He found that all were capable of work, that 7 were symptom-free, and that the remaining case had periodic pain.

CONCLUSIONS.

1. A series of 64 cases of acute perforated peptic ulcer is reported : 63 were treated by simple suture and 1 by suture plus gastro-enterostomy. The mortality-rate in cases operated on under twelve hours was 0·5 per cent, and in cases over twelve hours, 15·0 per cent.   The total mortality was 4·7 per cent.   We are of the opinion that these low figures fully justify the use of simple suture as the routine treatment of acute perforated ulcer.

2. The remote results of simple suture, suture plus gastro-enterostomy, and excision and pyloroplasty are not so good as those recorded after resection.   On this account we feel that in treating recurrent ulcers or complications, one should be prepared to consider partial resection rather than palliative measures as a secondary operation.

3. We found that approximately one-third of our cases eventually had to have a secondary operation performed. the remaining two-thirds being in such good health as to preclude the necessity for further operative interference.

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CARCINOMA OF THE CÆCUM.

A DISCUSSION OF ITS INCIDENCE, DIAGNOSIS, AND TREATMENT, WITH A REPORT OF TWENTY-FIVE PERSONAL CASES.

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If the rectum is excluded, 10 per cent of cases of cancer of the large intestine occur in the cæcum. During the period 1920–9 inclusive, 322 cases of carcinoma of the large intestine, other than those occurring in the rectum, were admitted to King's College Hospital ; of these 31 were cases of carcinoma of the cæcum. It might be thought that the percentage of cæcal cancers would be higher, considering the irritation to which the cæcum must be subject owing to the fluid contents of the small intestine being squirted through the ileocæcal valve and impinging upon its outer wall. On the other hand, the contents of the cæcum are normally fluid, and thus when once the cæcum is full, its mucosa is not nearly so liable to irritation as is that of the distal (left) part of the colon, where the contents are more solid, owing to the absorption of water which takes place chiefly in the proximal (right) part of the colon.

    Development of the Cæcum.—About the ninth week of intra-uterine life, the abdominal viscera begin to recede within the parietes, the fœtal peritoneal cavity being at this stage sufficiently large to contain all the abdominal viscera. The return of the umbilical loop of the intestine is accompanied by an important process of rotation of the intestinal tube around the axis of the superior mesenteric artery. The small intestine enters the peritoneal cavity below and to the left of the large intestine and its mesocolon. The upper part of the large intestine together with its mesocolon come into contact with the posterior wall of the omental bursa with which they fuse, and thus the transverse mesocolon is formed. The cæcum is, at an early stage, immediately below and in contact with the liver. At this stage the cæcum may be regarded as marking the right extremity of the transverse colon. The liver and cæcum eventually become separated one from the other. The separation is effected by three growth factors : (1) A relative (not actual) diminution in size of the liver—the liver may be regarded as shrinking headwards ; (2) A relatively rapid elongation of the trunk ; (3) An actual displacement caudalwards of the cæcum into the right iliac fossa—this displacement is of relatively small extent. As the liver and the cæcum become displaced, one from the other, the large gut distal to the cæcum elongates, and thus the ascending colon makes its appearance. Under certain but somewhat rare circumstances, the cæcum undergoes no relative or actual displacement from its original position,

but persists in contact with the liver and follows this viscus in its displacement headwards. In such cases no ascending colon exists. In other cases the actual migration downwards of the cæcum into the right iliac fossa fails to take place, and the cæcum occupies a higher position than is usually the case, and is found at some little distance above the iliac fossa and leads into a relatively short ascending colon. Sometimes rotation of the gut fails to occur. In such cases the colon occupies the left lower portion of the abdominal cavity, has no connection with the great omentum, and the cæcum is found on the left side of the abdomen. A very rare condition exists in which, owing to the adhesion of its mesentery to the posterior wall of the omental bursa in early fœtal life, the cæcum actually lies in the transverse mesocolon.

Such developmental abnormalities, the majority of which are fortunately rare, must be kept in mind, as carcinoma of the cæcum might be mistaken for carcinoma of some part of the ascending, transverse, or sigmoid colon, for which entirely different surgical treatment would be requisite.

Surgical Anatomy of the Cæcum.—The main points of importance are its: (1) Attachments ; (2) Blood-supply ; (3) Lymphatic drainage.

Attachments.—The cæcum is, as a rule, completely invested by peritoneum, and for this reason is a mobile viscus. Occasionally the postero-superior wall has no peritoneal investment, and is applied to the posterior abdominal wall to which it is fastened by areolar tissue. Generally speaking, therefore, the surgical mobilization of the cæcum may be said to be free from difficulty.

Blood-supply.—The blood-supply of the cæcum is derived from the artery to the mid-gut—namely, the superior mesenteric artery. The vessels concerned are the anterior and posterior cæcal arteries, branches of the ileocolic artery, a terminal derivative of the superior mesenteric. The supplying vessels are more easily seen if the cæcum is examined from behind. The posterior cæcal branch is the larger of the two, and gives off the artery to the appendix. It can be easily realized that if an attempt is made to ablate the cæcum alone, some of the collateral vessels are endangered. These are more particularly the ileal branch of the ileocolic artery, which supplies the terminal 8 in. of the ileum ; and the right colic artery, which is responsible for the vascularization of the ascending colon and part of the transverse colon.

In order to avoid the risk of gangrene, a resection for carcinoma of the cæcum must include not only the cæcum itself but also the terminal 6 or 8 in. of the ileum, the ascending colon, and the proximal 3 or 4 in. of the transverse colon. In actual practice an anastomosis is usually first established between the terminal ileum and the middle of the transverse colon. Ten days later the resection is effected.

Lymphatic System.—The lymphatic vessels of the cæcum correspond fairly accurately to the vascular channels. The lymphatic glands may be conveniently grouped as follows : (1) A local group comprising : (a) Anterior cæcal glands ; (b) Posterior cæcal glands. (2) An outlying system comprising : (a) Appendicular glands ; (b) Ileal glands ; (c) Right colic glands. The arrangement of these glands can be seen on examining *Fig.* 30. The outlying glands drain into the superior mesenteric group of the pre-aortic lymphatic glands. Any or all of these lymphatic glands may be involved in cases of carcinoma of the cæcum.

It has been estimated by Craig and MacCarty that the lymph nodes are invaded in 32 per cent of cases. This we consider a very generous computation. when it is remembered that the usual enlargement met with is due to a purely inflammatory reaction which subsides when the absorbing area is removed. Communications between the lymphatic vessels of the appendix and cæcum and those of the broad ligament and iliac lymphatic vessels have been described. More recent observations. however. show that there is, at any rate, no primary connection. Nevertheless, extension of carcinoma may take place along the subserous spaces. and secondarily involve the pelvic organs and lymphatic glands.

Etiology of Carcinoma of the Cæcum.—The youngest case of carcinoma of the cæcum is one reported by Chajutin. which occurred in a girl aged 14 years : the majority of cases. however. occur from the fifth decade onwards. In 100 cases collected from the Mayo Clinic by Craig and MacCarty the average age incidence was 49, the disease being twice as common in males as in females. In our own personal series there were 19 male and 6 female cases—a higher proportion of males than is shown by the American figures. With regard to the habit of the individual, constipation can hardly be looked upon as a factor in its causation, otherwise the condition would be more likely to occur in females than in males.

FIG. 30.—Drawing showing the lymphatic drainage of the cæcum and proximal portion of the colon.

It is a significant fact that carcinoma occurs commonly in those parts of the alimentary canal which are developed from the 'fore' and 'hind' gut. The small intestine, cæcum, ascending colon, and hepatic flexure, which are derived from the 'mid' gut, are far less commonly affected with the disease. Carcinoma in the small intestine is very rare, and accounts for 2 per cent or less of cases of cancer of the whole intestinal tract. The reaction of the contents of the stomach and the large bowel is acid, while that of the small intestine is alkaline. In the former cancer is common ; in the latter it is very rare. Some trauma, which may be regarded as a biochemical trauma. must affect the mucosa lining the outer wall of the cæcum as the alkaline juices of the small intestine are squirted through the ileocæcal valve and impinge upon it.

Can the problem of malignant disease be elucidated in terms of hydrogen-ion concentration ? Does not the painting of the skin of rats with tar or a constant application of crude acids favour the appearance of epithelioma ? Again, is not the application of radium a means of altering the reaction of the tissue juices *in vivo* with the disappearance of the neoplasm in some cases ? In the present state of our knowledge it is impossible to state the cause of the disease, but what better field could there be for speculative thought ?

Pathology.—Microscopically, cancer of the cæcum conforms to one of several histological types : (1) Adenocarcinoma ; (2) Fibrocarcinoma ; (3) Colloid carcinoma ; (4) Carcinoma arising in a polypus.

1. *Adenocarcinoma* in the cæcum is the commonest type, and does not differ materially from the same type arising in other parts of the intestinal tract. The acini are lined with typical columnar cells which stain deeply. The acini are irregular in size. Infiltration occurs into the submucosa and may extend into the muscle coat, but usually not deeply or widely.

2. *Fibrocarcinoma* undergoes ulceration in most cases. Fibrosis is variable, but may be very marked. The cells are atypical and are seen in small groups, infiltrating the muscular coat deeply. Infiltration is a marked feature of the growths.

3. *Colloid carcinoma*, better termed 'mucoid degeneration'. This occurs in the adenocarcinoma and all structure may be lost. Small groups of cells only may remain. The mucoid material stains with muci-carmine and exhibits a typical appearance.

4. *Carcinoma arising from polypi* are rare in the cæcum, but have been reported. The carcinoma is of the adenocarcinomatous type. Polypi may show a papillary form of growth. If mucoid degeneration has occurred, the diagnosis of malignancy may be very difficult.

The growth commences in the mucous membrane as an adenocarcinoma, and if we consider the outer wall of the cæcum opposite the ileocæcal valve as the commonest site, the growth first extends downwards to the caput cæci and later extends upwards from the caput cæci to the ileocæcal valve. Owing to the method in which the carcinoma spreads, the ileocæcal valve is not involved until a comparatively late date.

The majority of cases of carcinoma of the cæcum come up for treatment owing to the recognition of a lump in the right iliac fossa. In some cases the growth tends to project into the lumen of the cæcum as a large, fungating cauliflower-like mass (*see Fig.* 32), and in such cases septic absorption and toxæmia occur, and a leucocytosis may be present. In other cases, however, the growth infiltrates the wall, and causes, in some cases, a carcinomatosis of the peritoneum ; ultimately the liver may be involved in a carcinomatous capsule (*see Fig.* 39). Colloid degeneration may occur in any type of carcinoma of the cæcum, and is probably as common as in the stomach. Metastatic deposits in the liver from carcinoma of the cæcum may show the typical multiple, yet somewhat isolated, hard areas with umbilication which occur on the surface of the liver (*see Fig.* 35). On the other hand, the liver may be so infiltrated with secondary deposits that no normal liver-tissue can be seen with the naked eye. Again, the liver may be surrounded by a carcinomatous capsule and yet have no actual deposits within it (*see Fig.* 39).

Microscopically, carcinoma of the cæcum is a typical adenocarcinoma (*see Figs*. 34, 37), and frequently the inflammatory reaction due to the superficial septic condition of the tumour can be easily seen.

Symptomatology.—The symptoms are very variable and cannot be said to conform to any definite type. Perhaps the most constant feature is the presence of a palpable tumour. This, we are convinced, is due to the fact that the usual origin of the growth is on the outer wall of the cæcum, whence it extends downwards into the caput cæci, and thence upwards along the inner wall until the ileocæcal valve is more or less occluded. When this happens, symptoms of chronic obstruction arise. There is rarely any pain except in very advanced cases, but constipation is a common symptom. Some degree of anæmia is usually present. Intestinal obstruction is uncommon, except in very advanced cases where the ileocæcal valve is almost completely occluded. There is commonly tenderness in the right iliac fossa in the region of the palpable tumour, and for this reason the condition is sometimes diagnosed as appendicitis. A leucocytosis is present in about 50 per cent of cases and is undoubtedly due to septic absorption from the tumour in patients with good resistance ; the presence of leucocytosis is another factor which favours the diagnosis of appendicitis.

In a third of the recorded cases there is no history whatever of loss of weight. It is a remarkable fact that few cases ever complain of nausea. The average duration of symptoms before operation varies between nine months and two years. In our series the shortest period was three weeks, while the longest was three years. In five cases only was visible peristalsis present. This is a rather interesting fact, as it is stated in students' text-books that carcinoma of the cæcum is frequently attended by visible peristalsis of the terminal coils of the small intestine. Blood was noticed in the stools in three of our cases only.

Differential Diagnosis.—A correct diagnosis of carcinoma of the cæcum should be made in the majority of cases, as so few of them are masked by symptoms due to acute intestinal obstruction.

As a palpable swelling in the right iliac fossa is nearly always present, a differential diagnosis has to be made from ileocæcal tuberculosis, abscess of the appendix, and actinomycosis of the cæcum or appendix. Ileocæcal tuberculosis is a rare disease in the London area, and the age incidence is much lower than that of cancer of the cæcum. Loss of weight, anorexia, and passage of blood and mucus in the stools are marked and significant symptoms. Appendicular abscess is generally more painful and is not so resistant on palpation as a neoplasm of the cæcum. In actinomycosis of the cæcum the diagnosis is rarely made except in those cases in which a sinus or sinuses are present and yellow granules are seen in the pus.

Perhaps the most useful aid to differential diagnosis is an X-ray examination. By means of a barium meal or enema, a correct diagnosis can usually be made. In carcinoma of the cæcum the filling defect of the cæcum can usually be seen (*see Fig.* 36). In appendicular abscess the cæcal outline is usually normal. In ileocæcal tuberculosis the terminal ileum is involved as well as the cæcum and a thin narrow channel can be seen leading from the pelvis into a small contracted cæcum. In actinomycosis of the cæcum, although

the cæcal capacity is reduced by the thickening in its walls, there is no marked irregularity of the outline as is seen in cases of carcinoma of the cæcum.

Treatment.—There can be no doubt that carcinoma of the cæcum can only be treated satisfactorily by surgical means. X-ray and radium therapy are of little use. The surgical treatment demands a two-stage operation. Although excision of the growth and an ileocolostomy have been successfully performed at one operation in a few recorded cases, the correct procedure should be first to effect a lateral anastomosis between the terminal ileum and the middle of the transverse colon, and after an interval of a week or ten days, to excise the last 6 or 8 in. of the ileum, the cæcum, ascending colon, and hepatic flexure. There is often some difficulty in peritonizing the raw surface which is left on the posterior abdominal wall in the region of the hepatic flexure ; this raw surface, however, can easily be covered over with the omentum, which can be fixed there by a few interrupted sutures.

In one case only in our series was excision of the growth together with an ileocolostomy performed at one and the same operation. The patient died seven days later (*Case* 16). In cases complicated by intestinal obstruction, ileocolostomy should be performed, and the anastomosis should be reinforced by an omental graft to obviate the possibility of leakage through the suture holes in the distended small intestine.

CASE REPORTS.

Case 1.—Mr. A. J., age 58, was admitted to King's College Hospital, on July 11, 1915, complaining of slight pain in the abdomen, which his doctor thought was due to appendicitis. The patient had complained of slight nausea for some time, but had vomited on only two occasions, the day prior to his admission to hospital. There was no history of constipation, and the man had always enjoyed good health until three weeks before coming to hospital.

ON EXAMINATION.—The patient was found to be a well-built and muscular man, with a furred tongue and a temperature of 90°. The abdomen was distended, there was marked tenderness in the right iliac fossa, and a definite lump could be easily felt in that situation. A leucocyte count revealed 22,000 leucocytes per c.mm. A diagnosis of appendix abscess was made, and a laparotomy was done six hours after admission.

OPERATION.—When the patient was well under the anæsthetic it was found that the lump in the right iliac fossa was freely movable and not fixed. For this reason a paramedian incision was made and the rectus muscle displaced outwards. A large cæcal tumour was seen, an anastomosis between the ileum and the transverse colon was performed, and the wound closed. Twelve days later the growth was excised, including the terminal 6 in. of the ileum, the ascending colon, and the hepatic flexure. The patient made an uninterrupted recovery, and left hospital on Aug. 10. He reported himself as quite fit and well in December, 1930, which is more than fifteen years since his operation.

The specimen (*Fig.* 31) shows a large nodular growth which appears to have originated on the outer wall of the cæcum and grown downwards towards its base. There was no involvement of the ileocæcal valve. Histologically the growth was a typical adenocarcinoma.

Case 2.—Mrs. F. W., age 65, was admitted to hospital on Sept. 18, 1915, with acute intestinal obstruction of three days' duration. Her husband stated that she had suffered from severe constipation on and off for four years and had lost a considerable amount of weight during the last year or so. Her bowels had not acted for six days previous to admission to hospital.

ON EXAMINATION.—Her condition was poor, the abdomen was greatly distended with generalized tenderness. No tumour could be felt and the rectum was empty. An enema was returned clear.

OPERATION.—Under light ether anæsthesia a paramedian incision was made, and a large carcinoma of the cæcum was found. There was considerable ascites, and secondary deposits in the liver were felt. The ileum was quickly anastomosed to the transverse colon, and the abdomen closed. The patient died eighteen hours after the operation.

AUTOPSY.—The growth was found to involve the whole cæcum and had occluded the ileocæcal valve. There was extensive glandular involvement in the ileocæcal angle and along the posterior abdominal wall. The liver was studded with secondary deposits. The growth was a typical adenocarcinoma.
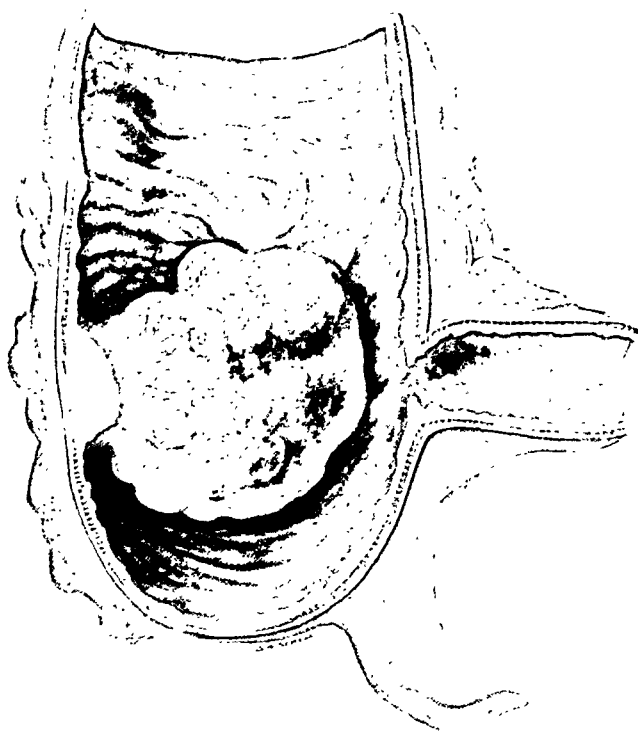
FIG. 31.—*Case* 1.   Carcinoma of the cæcum involving the outer wall. The growth exerts no effect on the ileocæcal valve.

Case 3.—Mr. J. A. T., age 62, was admitted to hospital on Dec. 11, 1915, complaining of pain in the abdomen which was intermittent in character, and was first noticed about six months previously.

ON EXAMINATION.—A hard tumour could be easily palpated in the right iliac fossa. There was no abdominal distension, and only slight pain on palpation. The pulse-rate and temperature were normal. The leucocyte count was 8200 per c.mm. An enema produced a good result. The day following admission an operation was performed.

OPERATION.—A right paramedian incision was used to explore the abdomen. A hard growth about the size of a bantam's egg was felt in the cæcum. The rest of the abdomen appeared to be normal, and there was no evidence of any glandular

involvement. The terminal ileum was anastomosed to the middle of the transverse colon, and the abdomen closed. Thirteen days later the growth together with the terminal 6 in. of ileum, the ascending colon, and hepatic flexure were removed. The patient made an uninterrupted recovery. The growth was nodular and somewhat circumscribed, and histologically it was an adenocarcinoma.

We ascertained that this patient died in 1927 from pneumonia. There was no evidence of any recurrence during the twelve years following the operation.

Case 4.—Mr. H. W., age 56, was admitted to hospital on Jan. 11, 1916, complaining of piles.
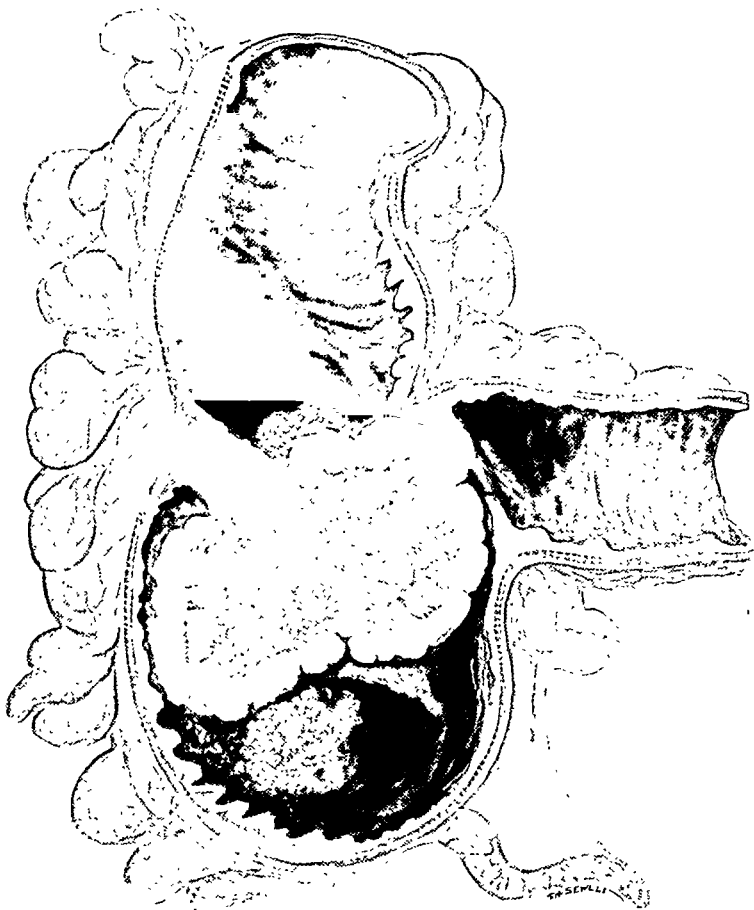
Fig. 32.—*Case 4.* Large polypoid carcinoma of the cæcum. The growth is attached to the outer wall of the cæcum.

ON EXAMINATION.—During a routine examination of the abdomen, a very definite lump was felt in the right iliac fossa. The lump was hard, slightly painful on palpation, but freely movable. On examination of the rectum nothing abnormal was felt or seen, with the exception of three internal piles. A leucocyte count revealed 7600 leucocytes per c.mm. A laparotomy was advised, and on Jan. 15 a right paramedian incision was made, and the abdomen explored. A large growth was felt involving the whole of the cæcum. There was no evidence of glandular involvement or ascites.

OPERATION.—An ileocolostomy was performed. Eleven days later the cæcum, ascending colon, and hepatic flexure were removed. The patient made an excellent recovery.

The growth (*Fig.* 32) was pedunculated, and its pedicle was just opposite the ileocæcal valve. It appeared to fill the whole cæcum, and was just commencing to press on the ileocæcal valve. Histologically it was an adenocarcinoma. The wife of this patient wrote in 1926, saying that her husband had just died from uræmia. He had lived ten years after his operation without any sign of recurrence.

Case 5.—Mr. J. B., age 64, was admitted to hospital on May 5, 1916, as a case of acute appendicitis.

ON EXAMINATION.—The abdomen was distended and painful on palpation. A small hard lump could be felt in the right iliac fossa. The temperature was normal, and the pulse-rate 80. During the twelve hours previous to admission the patient had vomited frequently, and there was a history of melæna. During the last four days there had been no action of the bowels. The patient stated that he had suffered from pains in the abdomen and constipation for the last two years.

OPERATION (May 5).—The abdomen was opened by a right paramedian incision. An extensive carcinoma of the cæcum was exposed. There were numerous enlarged glands along the posterior abdominal wall and a few secondary deposits in the liver. There was a little free fluid in the abdomen. An anastomosis between the terminal ileum and the middle of the transverse colon was performed and the abdomen closed.

The man made an excellent recovery, and was discharged three weeks after the operation. He became jaundiced in August, 1916, and died in September, just four months after the operation. Unfortunately, no histopathology was available in this case.
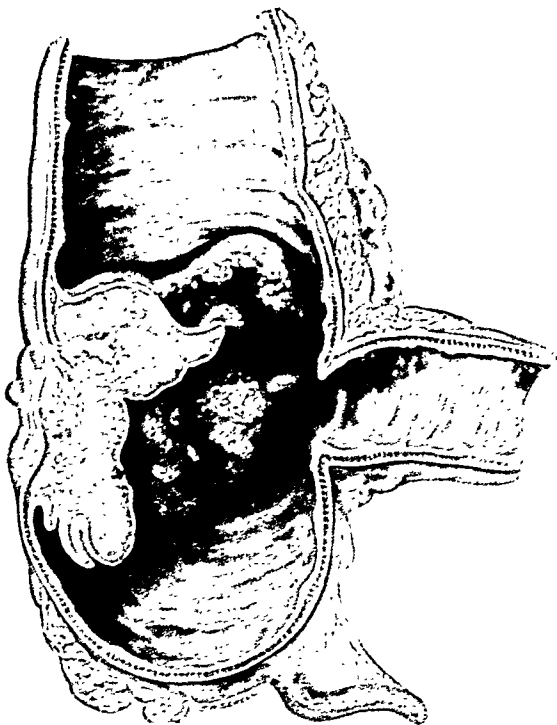
FIG. 33.—*Case 6.* Moderate-sized cancerous growth commencing on the outer cæcal wall.

Case 6.—Mrs. A. T., age 32, was admitted to hospital on Aug. 4, 1916, as a case of acute appendicitis. She had suffered from intermittent pain in the abdomen for about five months, but during the last two days the pain had become worse, and was localized in the right iliac fossa.

ON EXAMINATION.—The abdomen was not distended. A definite tumour could be felt in the right iliac fossa. There was a leucocytosis of 10,270 per c.mm. The diagnosis of acute appendicitis appeared to be correct.

OPERATION.—A right paramedian incision was made and the abdomen explored ; the appendix was quite normal, and was found hanging over the brim of the pelvis.

A hard tumour could be felt in the cæcum about the size of a tangerine orange. The terminal ileum was anastomosed to the middle of the transverse colon, and the abdomen closed. Nine days later the *cæcum, ascending colon, and hepatic flexure* were removed. The patient made a quick recovery, and left hospital sixteen days after her second operation.

The growth (*Fig. 33*) had started on the outer wall of the cæcum opposite the ileocæcal valve and had extended into the cæcum. There was no obstruction to the ileocæcal valve. Microscopically the growth was an adenocarcinoma.

The patient was alive and well in December, 1930, some fourteen years after the removal of the cancer. This is the youngest patient on whom we have excised a cancer of the cæcum.

Case 7.—Mr. J. T., age 61, came under observation early in 1917 suffering from indigestion and slight loss of weight. A barium meal examination was made on H.M. Hospital Ship *Garth Castle* by Surgeon-Commander Rooney, and a very definite irregularity was seen in the cæcum. The patient gave a history of vague dyspepsia for some two and a half years, but this might have been due to the strain of war-time in the R.N.R.

ON EXAMINATION.—The patient was found to be a well-built man who appeared to have lost some weight. The examination of the abdomen was negative except for the presence of a hard movable tumour in the right iliac fossa. The leucocyte count was 8164 cells per c.mm.

OPERATION (April 2, 1917).—Laparotomy was performed under ether anæsthesia, and a large cæcal growth was found. An ileocolostomy was performed between the terminal ileum and the transverse colon. Ten days later the cæcum, ascending colon, and hepatic flexure were excised. There were several glands in the ileocæcal angle ; these were removed and on section were found to contain growth.

The patient made a good recovery, but he was invalided out of the service, and died in 1919. He developed an enormous liver and ascites before death.

Case 8.—Mr. W. S., age 58, was admitted to hospital in June, 1918, with a diagnosis of acute appendicitis. He had suffered from slight pain in the abdomen and some constipation for nine months previous to admission.

ON EXAMINATION.—The patient looked somewhat anæmic and had a furred tongue. The abdomen moved well on respiration ; there was slight pain on pressure in the right iliac fossa and a small lump in that situation could be distinguished with difficulty. The temperature was 99° and the pulse-rate 80. Rectal examination simply revealed a loaded rectum. A simple enema produced a large constipated stool. Leucocyte count was 5602 per c.mm. The day following admission the patient had lost practically all his pain, and his temperature was normal. The lump in the right iliac fossa, however, was still present. Operation was advised.

OPERATION (June 4).—A laparotomy was performed through a right paramedian incision. A growth in the cæcum was discovered, and an ileocolostomy was performed. Ten days later the abdomen was again opened, and the terminal ileum, cæcum, ascending colon, and hepatic flexure were excised. There were no enlarged glands and no secondary growths in the liver. The patient made an uneventful recovery.

The histology of the growth proved it to be an adenocarcinoma. The patient reported himself every six months until 1928, when his wife wrote and said he had died from pneumonia, which caused his death in four days. He had remained quite fit from the time of his operation until his death, some ten years later.

Case 9.—Mr. H. B., age 62, was admitted to hospital on May 6, 1919, with acute intestinal obstruction. He gave a history of abdominal pain for the last two years and marked loss of weight. Constipation had troubled him for years, but for the four days previous to admission he had not been able to get his bowels open even with castor oil. He was vomiting continuously.

ON ADMISSION.—The patient was in a collapsed state, with a much distended abdomen. An enema only produced a little flatus. Although the abdomen was

greatly distended, a tumour could be palpated in the right iliac fossa. A blood-count revealed a definite anæmia, the leucocytes being 7460 per c.mm. The patient was kept warm in bed for several hours and his stomach was washed out.

OPERATION.—This was performed six hours after admission. A paramedian incision was used, and the abdomen opened. There was a considerable amount of ascites ; a large cæcal carcinoma was exposed ; it was fixed and inoperable. The terminal ileum was anastomosed to the middle of the transverse colon. Secondary deposits were felt in the liver. The patient had a prolonged convalescence, and left hospital six weeks after his operation. After being at home for two months, however, he was re-admitted to hospital because of ascites and some pain in the spine. He died on Aug. 10, 1919, just five months after his operation.

AUTOPSY.—This revealed generalized carcinomatosis, secondary deposits in the liver, lungs, and vertebral column. The growth was an adenocarcinoma.

FIG. 34.—*Case* 11. Microscopical drawing of the growth removed. (× 65.)

Case 10.—Mr. R. T., age 54, came under observation in January, 1920, com-plaining of vague pains in the abdomen. He had always enjoyed good health, and worked as a butcher.

ON EXAMINATION.—Examination in the out-patient department did not reveal anything abnormal. As his pain did not disappear after purgation, a barium-meal examination was made and this revealed definite irregularity in the cæcum. After this it was just possible to make out a cæcal tumour on deep palpation. There was no history of loss of weight or of vomiting or of melæna. A blood-count showed slight anæmia with 5664 leucocytes per c.mm.

OPERATION (Feb. 2).—The abdomen was opened through a right paramedian incision. A small carcinoma of the cæcum was found, and an ileocolostomy was performed. Ten days later the growth, ascending colon, and hepatic flexure were excised. The patient made an excellent recovery, and returned to work six weeks after his operation. The growth was limited to the outer wall of the cæcum, and was an adenocarcinoma. The patient was alive and well in December, 1930—ten years after his operation.

Case 11.—Mr. G. H., age 58, was sent up to hospital in April, 1920, diagnosed as a case of acute appendicitis. His history was one of vague dyspepsia and abdominal pain for ten months, which had suddenly become worse.

ON ADMISSION (April 14).—The temperature was 100° and the pulse 96. There was slight tenderness in the right iliac fossa, and a lump could just be palpated; otherwise the signs were completely negative. The leucocyte count was 6400 cells per c.mm.

OPERATION.—Five hours after admission operation was performed. Under the anæsthetic the lump in the right iliac fossa could be easily palpated; it was found to be fixed to the cæcum, but freely movable with the cæcum on the posterior abdominal wall. A right paramedian incision was used, and a carcinoma of the cæcum was exposed and brought out of the wound. As the case was an early one and the patient's condition good, we were almost tempted to excise the tumour and do an anastomosis at one and the same time. Better judgement prevailed, however, and an ileocolostomy was performed. Seven days later the growth was excised, together with the terminal ileum, ascending colon, and hepatic flexure. The patient made an uneventful recovery.

The growth had started on the postero-external wall of the cæcum and grown towards the ileocæcal valve, and was just beginning to compress this. The caput cæci was not involved at all. There were no glands in the ileocæcal angle. The tumour was a typical adenocarcinoma (*Fig.* 34). This patient was alive and well in December, 1930—some ten years after the operation.

Case 12.—Emma Y., age 64, was admitted to hospital on Sept. 19, 1920, with acute intestinal obstruction. Her history was that for the last twenty-one months she had suffered from increasing constipation, culminating in complete cessation of motions six days before admission. The day prior to admission the patient commenced to vomit, and this increased until it became almost constant.

ON EXAMINATION.—The woman looked extremely ill, with a rapid pulse and a temperature of 97·8°. Her abdomen was greatly distended and tender, and no lumps or tumour could be palpated. Rectal examination simply revealed a ballooned condition of the bowel. An enema was given and returned clear. The stomach was washed out with some sodium bicarbonate solution and a pint of subcutaneous saline was given. The condition of the patient improved somewhat, and six hours after admission operation was decided upon.

FIG. 35.—*Case* 12. Secondary deposits in the liver. There is more carcinomatous tissue than liver tissue.

OPERATION.—Under gas and oxygen anæsthesia the abdomen was opened by a right paramedian incision. There was some ascites and secondary deposits in the liver. The primary growth was a large one and situated in the cæcum; it had almost completely occluded the ileocæcal valve. A rapid ileocolostomy was performed, and two pints of 6 per cent gum acacia were given intravenously. The patient recovered from the operation and her bowels acted normally, but the liver became very enlarged and the ascites increased. She died of hepatic insufficiency just two months after her operation.

AUTOPSY.—There was found to be considerable glandular involvement in the

para-aortic glands. The liver was studded with large umbilicated secondary growths (*Fig.* 35). There were also secondary growths in the lungs and three small growths in the brain. The operation relieved her pain and made her comfortable for the remainder of her existence.

Case 13.—Mr. C. A., age 56, first attended hospital as an out-patient in December, 1920, as his doctor had told him that he was suffering from a gastric ulcer.

ON EXAMINATION.—Clinical examination did not reveal anything of import except slight tenderness in the right iliac fossa, and the question whether or not this case was one of appendicular gastralgia was raised. A barium meal examination was made, and this showed a normal stomach and duodenum, but a very definite cæcal irregularity (*Fig.* 36). In spite of this examination no definite tumour could be palpated.

OPERATION.—This was advised on the X-ray findings, and the abdomen was opened by a paramedian incision on Jan. 3, 1921. A small carcinoma of the cæcum was discovered, and an ileocolostomy performed. Ten days later the growth and ascending colon and hepatic flexure were excised. There was no sign of secondary growths in the liver or elsewhere. The patient left the hospital three weeks after his second operation. The growth was a typical adenocarcinoma.

The patient was alive and well in December, 1930, with no sign of any recurrence whatsoever. It has often been stated that much time and material are wasted by patients taking unnecessary barium meals. In this case, however, it was due to the X-ray examination alone that the diagnosis was made at an early stage. A successful operation was performed as the result.

Case 14.—Mr. J. M., age 52, was sent up to hospital on March 4, 1921, with a diagnosis of acute appendicitis. He had suffered from vague abdominal pains and slight constipation for eight months. On

FIG. 36.—*Case* 13. Skiagram showing the filling defect in the cæcum. The amount of barium in the cæcum is very small.

the day of admission, as he felt 'off colour' and did not want his food, he visited his panel doctor, who sent him to the hospital.

ON EXAMINATION.—He did not look very ill; his tongue was furred and his temperature 99°. The abdomen was not distended, but there was slight tenderness in the right iliac fossa, and a lump about the size of a pigeon's egg could be indistinctly palpated. Rectal examination simply revealed a loaded bowel. Leucocyte count showed 5400 cells per c.mm.

OPERATION.—Under the anæsthetic the tumour in the right iliac fossa became very definite and was found to be freely movable. A right paramedian incision was used to open the abdomen. A small carcinoma of the cæcum was discovered and an ileocolostomy performed. Ten days later the growth, together with the ascending colon and hepatic flexure, was excised. The patient left hospital eighteen days after his second operation; he was alive and well in December, 1930. The only complaint that this patient has, is that his stools are inclined to be loose, and he sometimes has three or four actions a day.

The growth, which was situated on the outer wall of the cæcum, was an adenocarcinoma (*Fig.* 37). There was no evidence of any glandular involvement at the time of the operations.

Case 15.—Mr. T. W., age 58, was admitted to hospital in April, 1922, suffering from chronic obstruction. He had suffered from indigestion for the last eighteen months and this had recently become worse. He now suffered from nausea and occasional vomiting attacks. He had lost weight and was becoming weaker every month.

ON EXAMINATION.—The man looked definitely ill and anæmic. The abdomen was distended, and a large hard fixed tumour could be felt in the right iliac fossa. Rectal examination was negative. A blood-count revealed a definite anæmia and 6522 leucocytes per c.mm.

FIG. 37.—*Case* 14. Microscopical drawing of the growth removed. (× 65.)

OPERATION (April 8).—The abdomen was opened by a right paramedian incision and a very large carcinoma of the cæcum exposed. The growth was hard and fixed, and removal was impossible. An ileocolostomy was performed between the terminal ileum and the middle of the transverse colon. There were numerous hard glands on the posterior abdominal wall, but no secondary deposits were seen on the surface of the liver. The patient recovered from his operation very well, and left hospital just over three weeks from the time of his operation. He was re-admitted, however, six months later with ascites and an enormously enlarged liver. He remained in hospital for six weeks and then died.

AUTOPSY.—There were numerous secondary deposits in the liver and lungs. The primary growth in the cæcum did not appear any larger than it was at the time of the operation, eight months earlier. On microscopical examination the growth proved to be an adenocarcinoma.

Case 16.—Mrs. E. F., age 62, was admitted to hospital on July 2, 1923, suffering from pain in the right iliac fossa and constipation. Two months previously she herself had noticed a lump on the right side of her belly. There was no vomiting and the patient's general condition was good.

ON EXAMINATION.—A large, hard, movable tumour could easily be palpated in the right iliac fossa. The liver was not enlarged and there was no sign of ascites. A blood examination revealed some anæmia and 7500 leucocytes per c.mm.

OPERATION (July 3).—The abdomen was opened through a right paramedian incision. A large carcinoma of the cæcum was exposed. The growth appeared to involve the whole cæcum and was beginning to press on the ileocæcal valve. The condition of the patient appeared good and there was no sign of secondary deposits. The growth and last 6 in. of ileum, ascending colon, and hepatic flexure were excised, and the ileum was anastomosed to the transverse colon. The patient suffered from severe shock after the operation, but had her bowels open on the third day and appeared to be going on well. On the seventh day after operation, however, her heart became dilated and she died.

The growth (*Fig.* 38) involved the whole cæcum, and had invaded the cæcal wall in many places. Histologically it was an adenocarcinoma. This patient would in all probability have lived if a two-stage operation had been carried out.

FIG. 38.—*Case* 16. Carcinoma involving the whole cæcum and the ileocæcal valve.

Case 17.—Mr. J. T., age 68, was admitted to hospital in November, 1923, his doctor having noticed a lump in his abdomen. He had complained of pain in the abdomen for about a year, and had suffered from increasing constipation. He stated that he had become weaker during the last few months and had lost weight.

ON EXAMINATION.—The abdomen was slightly distended, and a large, hard tumour could be felt in the right iliac fossa. The liver was slightly enlarged, but there were no signs of ascites.

OPERATION (Nov. 12).—The abdomen was opened through a right paramedian incision. A large, fixed carcinoma of the cæcum was exposed with some secondary deposits in the liver. Owing to the fact that the ileocæcal valve appeared to be partially blocked by growth, it was thought necessary to do a short-circuiting operation. The terminal ileum was anastomosed to the transverse colon. The patient recovered from the operation, but did not regain his strength. A month after the

operation there was definite enlargement of the liver and ascites. The patient gradually became weaker and died just two months after his operation.

AUTOPSY.—The liver was found to be three times its normal size and riddled with growth. There were also secondary growths in the lungs. The peritoneum and intestines were studded with secondary nodules. The growth, histologically, was an adenocarcinoma.

Case 18.—Mr. F. K., age 56, came under observation in March, 1924, suffering from hæmorrhoids, and as these did not disappear with the application of lotions and ointments, he was admitted for operation.

ON EXAMINATION.—On routine examination prior to operation a lump was palpated in the right iliac fossa. A leucocyte count was taken, and revealed 7480 cells per c.mm. With the exception of three internal piles the rectal examination was negative. The lump in the iliac fossa did not disappear after purgation, it underwent no change, and was freely movable. The condition was diagnosed as carcinoma of the cæcum, and operation was advised.

OPERATION (April 2).—Laparotomy was performed through a right paramedian incision. A carcinoma of the cæcum was exposed, and an ileocolostomy between the terminal ileum and the transverse colon was performed. Ten days later the growth and the right colon were excised. There was no evidence of any secondary deposits. The patient was able to go home eighteen days after his second operation. The growth was about the size of a tangerine and was limited to the outer wall of the cæcum opposite the ileocæcal valve. There were no enlarged glands in the ileocæcal angle.

Histologically the growth was a typical adenocarcinoma. The patient was alive and well in December, 1930—over seven years after the removal of the growth.

Case 19.—Mr. E. T., age 61, was admitted to hospital on June 4, 1925, with a diagnosis of acute appendicitis. He stated that his bowels had been troublesome for the last eighteen months.

ON EXAMINATION.—His temperature was 90°, pulse 80, respiration rate 20, and the tongue furred. Abdomen moved well on respiration. There was some tenderness in the right iliac fossa and a definite lump about the size of a duck's egg could be palpated. Leucocyte count was 8000 per c.mm. Rectal examination was negative.

OPERATION.—This was performed six hours after admission. Under the anæsthetic the lump in the right iliac fossa was found to be movable. A right paramedian incision was used to open the abdomen. A carcinoma of the cæcum was exposed, and an ileocolostomy performed. On June 14 a second operation was performed, and the growth and the right half of the colon were excised. There was no evidence of any secondary growths. The tumour involved the outer wall and head of the cæcum ; the glands in the ileocæcal angle were enlarged, but did not contain any growth. The patient made an uneventful recovery. The growth was an adenocarcinoma.

The patient was alive and well in December, 1930—more than five years after the operation.

Case 20.—This case was reported by Dr. Anson, a former house surgeon to one of us (C. P. G. W.) in the *Lancet* (1927, i, 704).

A man of 65 was admitted to hospital on Aug. 18, 1926, with acute intestinal obstruction. The acute symptoms were relieved on admission by an asafœtida enema. The patient gave a history of a dull pain in the hypogastrium for three weeks. He had also been getting constipated, the motions becoming smaller in quantity and very hard, though of normal shape and colour. There had been no diarrhœa and no blood or mucus in the stools. Defæcation had become increasingly difficult, with much tenesmus. Micturition was normal, except that the quantity of urine had diminished slightly three days after the onset of constipation.

ON EXAMINATION.—The pulse-rate was 84, the temperature 98·4°. The abdomen was distended and prominent ; the movements were normal, and there was no rigidity and no tenderness. The hypogastrium was dull to percussion, with shifting

dullness in both flanks. The liver, spleen, and kidneys were not palpable. On rectal examination a hard nodular mass about the size of a large egg was felt in the anterior wall of the rectum 2 in. above the external sphincter. It was not movable, and the surface felt as if it was ulcerated, but no bleeding occurred on examination. A diagnosis of carcinoma of the rectum was made, and as obstructive symptoms increased, a colostomy was performed on Aug. 20.

OPERATION.—A paramedian incision 3 in. long was made through the left rectus muscle, and the peritoneum was opened. A condition of diffuse carcinomatosis was found, with small nodules over the small gut and colon, and also in the mesentery. About three pints of dark-brown ascitic fluid was drawn off, and a colostomy performed. The gut was opened, a Paul's tube was tied in, the wound was then sutured and packed round with iodoform gauze and a powder of boric and salicylic acids to prevent ascitic oozing.

On the day after the operation the patient vomited 30 oz. of foul coffee-grounds vomit. There was a slight action of the colostomy; although purgatives were given freely, an action of the colostomy became increasingly difficult. The liver and spleen became palpable on Oct. 4, and the patient died on Oct. 23.

AUTOPSY.—At autopsy the body was greatly emaciated; both lungs showed collapse and bronchopneumonia at the bases. Heart muscle showed brown atrophy. The peritoneal cavity was almost obliterated by adhesions and carcinomatous growth : there were numerous small nodules on the abdominal wall and over the surface of the gut. A huge mass of carcinoma was found beneath the left lobe of the liver, slightly adherent to it and to the stomach, but not invading the stomach wall. On examining the mass felt per rectum, it was found to be a secondary deposit situated between the rectum and bladder. As it was obviously not the primary growth, the viscera were removed *in toto* with part of the posterior abdominal wall : they were hardened in 10 per cent formalin, and a detailed dissection was subsequently carried out.

The dissection showed a large, soft, fungating mass of carcinoma of the typical cauliflower variety filling the cæcum and occluding the ileocæcal valve. The large mass of growth already referred to as lying below the left lobe of the liver was found to be a huge secondary deposit in the pre-aortic glands. Spreading out from it were numerous nodules in the mesentery which were almost confluent at its base, but became more aparsely scattered as the free margin was approached. The splenic hilum was invaded by growth spreading from the region of the cœliac group of pre-aortic glands ; the periphery of the liver was invaded by a mass of growth which completely encapsuled the organ, the enclosed liver tissue being perfectly normal (*Fig.* 39). The duodenum and stomach were normal, although part of the greater curvature was adherent to the spleen and to the mass of growth springing from the pre-aortic glands. The pancreas, kidneys, and prostate were normal.

Microscopic examination of the primary growth in the cæcum showed a typical adenocarcinoma with very well-marked colloid degeneration. The secondary deposits in the peritoneum over the small gut and colon and invading the periphery of the liver were of similar structure, as was also the mass lying between the rectum and bladder. This deposit of growth had penetrated the muscular coats of the rectum and invaded the mucosa and was obviously just about to ulcerate in the lumen of the rectum.

This case offers an interesting example of mis-diagnosis, the error being chiefly due to the unusual position of part of the secondary deposit—namely, between the rectum and the bladder. In the light of the above findings it appears that doubts might have been raised as to the correctness of the diagnosis by the continuation of some degree of obstruction after colostomy, and also by the evidence of extensive secondary deposits, although the primary seat of growth in the rectum had not yet ulcerated through into the lumen of the bowel. On the other hand, in the carcinoma of the cæcum secondary deposits are usually late in appearance, and, moreover, relief of obstruction occurring at the ileocæcal valve would not be expected to follow the operation of colostomy.

There seems little doubt that in this case the original attack of acute obstruction relieved by colostomy was due to the combined action of the growth obstructing

the ileocæcal valve, and the secondary mass narrowing the lumen of the rectum below. The failure of the colostomy to act freely and the subsequent increasing obstruction were due to the primary seat of growth obliterating the ileocæcal valve more and more completely, until finally almost complete obstruction again resulted. Another unusual feature is the fact that there was a comparatively early and typical primary growth in the cæcum and very extensive secondary deposits all over the peritoneal cavity.

The case presents rather a striking example of the spread of the carcinoma by at least two well-defined and separate paths—namely, peritoneal and lymphatic spread. The peritoneal spread is shown by the presence of small nodules in the peritoneum of both the anterior and posterior abdominal walls, while the secondary

FIG. 39.—Case 20. Secondary carcinomatous capsule surrounding the liver. There were no growths in the substance of the liver.

nodules situated on the surface of the small gut were also seen in the peritoneum. Microscopic examination showed that only the outer portion of the muscular coats of the gut had been invaded, the mucosa being quite intact. A peritoneal spread, moreover, offers the only satisfactory explanation of the rare phenomenon of a complete carcinomatous capsule surrounding the liver and infiltrating its periphery for about an inch, while the central portion was left completely normal with no trace of any secondary deposit in its substance. The mass lying in the rectovesical

pouch which lead to the error in diagnosis was due either to direct peritoneal spread or to a small mass of carcinoma cells becoming detached into the peritoneal cavity and then falling into the rectovesical pouch, there undergoing development. The lymphatic spread is shown by enlargement of the ileocolic groups of glands, which could be traced upwards until the huge secondary mass lying in the superior mesenteric group of pre-aortic glands was reached. Further lymphatic spread from this point along the superior mesenteric afferent lymphatics as shown by the nodules of growth becoming more sparsely scattered through the mesentery as its periphery was approached ; while at its base they formed a large confluent mass of carcinoma as already described. The deposit at the splenic hilum was due to the permeation of growth from the superior mesenteric group to the cœliac group of pre-aortic glands and thence along the afferent vessels to the splenic hilum.

It must also be mentioned that two small secondary deposits of growth were found in the sternum, opposite the 7th left and 3rd right ribs respectively. This would appear to indicate some dissemination of the growth by the blood-stream, although no further evidence of such a path of spread could be found.

Case 21.—Mr. R. W., age 54, was admitted to hospital on Feb. 3, 1927, as a case of acute appendicitis. He had suffered from vague abdominal pains on and off for eight months prior to admission.

ON ADMISSION.—The temperature was 99·2°, pulse 88, and respirations 20. The abdomen was slightly distended, and a lump could easily be palpated in the right iliac fossa. Rectal examination was negative. Leucocyte count was 9500 cells per c.mm.

OPERATION (Feb. 3).—A right paramedian incision was used to explore the abdomen. A carcinoma of the cæcum was found, and an ileocolostomy performed. Nine days later the growth and right side of the colon were excised. No secondary deposits were seen in the abdomen. The patient left hospital three weeks after his second operation.

Histologically the growth was an adenocarcinoma. The patient was alive and well in December, 1930—nearly four years after his operation.

Case 22.—Mrs. A. B., age 59, was admitted to hospital on Aug. 6, 1927, with acute intestinal obstruction. She had always suffered from constipation, which had become more marked for the last two years. For four days previous to admission the constipation had been absolute and was accompanied by several attacks of vomiting.

ON EXAMINATION.—The patient was found to be very wasted and anæmic. The abdomen was distended, but in spite of this a tumour could just be felt in the right iliac fossa. Rectal examination was negative. A turpentine enema was returned clear.

OPERATION.—Laparotomy was performed the same day under gas and oxygen anæsthesia. The mass in the iliac fossa was found to be a large carcinoma of the cæcum involving the ileocæcal valve. The ileum was anastomosed to the transverse colon. Secondary deposits were felt in the liver, and there was free fluid in the peritoneal cavity. The patient recovered from the operation, but the liver increased markedly in size, and she died six weeks after the operation.

AUTOPSY.—There was generalized carcinomatosis of the peritoneum with multiple secondary growths in the liver. The growth was an adenocarcinoma with marked colloid degeneration.

Case 23.—Mr. J. A., age 55, was admitted to hospital on Jan. 2, 1928, as a case of acute appendicitis. He had suffered from pain in the right iliac fossa on and off for ten months, but on admission the pain had disappeared.

ON EXAMINATION.—Temperature 100°, pulse 90, respirations 22. There was some tenderness in the right iliac fossa, and on rectal examination no tumour could be felt. Leucocyte count was 6450 cells per c.mm.

OPERATION.—This was performed through a muscle-split incision ; the appendix was inflamed and a small growth was felt in the cæcum. The wound was enlarged

and an ileocolostomy performed. Ten days later the abdomen was opened through a paramedian incision and the cæcum and right half of the colon were excised. The growth was limited to the outer wall of the cæcum, just opposite the ileocæcal valve. There were no secondary deposits inside the abdomen. The patient made an uninterrupted recovery, and was alive and well in December, 1930—just three years after his operation.

Histologically the growth was an adenocarcinoma.

Case 24.—Mr. W. B., age 62, was admitted to hospital, a tumour having been discovered in his abdomen in the out-patient department. He had suffered from constipation for nearly two years, and had been under treatment for this for six months. He had lost a considerable amount of weight during the last year. There was a history of nausea but no actual vomiting
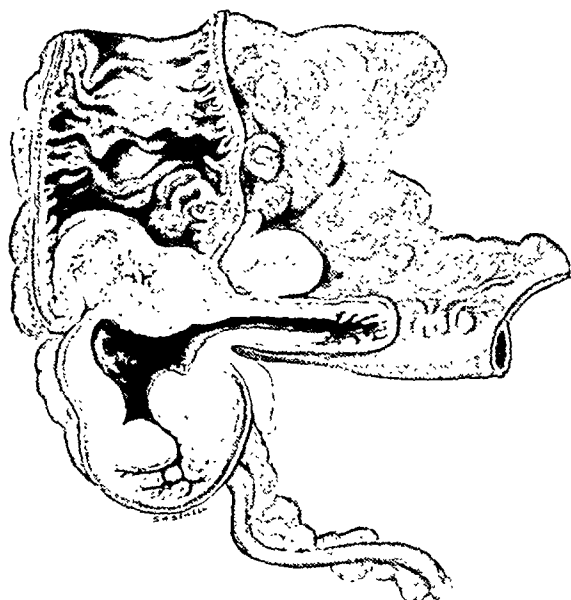
Fig. 40.—*Case 24.* Carcinoma of the cæcum growing into the ileocæcal valve.

ON EXAMINATION.—There was some abdominal distension and a lump could just be palpated in the right iliac fossa. Rectal examination was negative. Leucocyte count showed 8000 cells per c.mm.

OPERATION (Feb. 4, 1928).—Laparotomy was performed, and a carcinoma of the cæcum involving the whole cæcum and compressing the ileocæcal valve was exposed. The ileum was anastomosed to the transverse colon. Eleven days later the carcinoma together with the right half of the colon was removed. The patient made an uneventful recovery, and was alive and well in December, 1930—almost three years after the removal of the growth.

The tumour (*Fig.* 40) involved the whole cæcum and was growing into the ileocæcal valve. Histologically it was an adenocarcinoma.

Case 25.—Mrs. H. W., age 69, was admitted to hospital early in January, 1931, complaining of abdominal pain. She had enjoyed good health until six months previous to admission, when she first suffered from nausea. Her bowels had been somewhat costive of late. She was in the habit of taking salts every morning.

ON EXAMINATION.—The patient did not look her age, and seemed quite cheerful. The abdomen was slightly distended, and a hard lump, the size of a cricket ball, could

be palpated in the right iliac fossa. Rectal examination was negative. Leucocyte count showed 11,000 cells per c.mm.

OPERATION (Jan. 7).—The abdomen was opened through a right paramedian incision and a large fixed carcinoma of the cæcum exposed. There were many enlarged hard glands in the ileocæcal angle and on the posterior abdominal wall. An ileocolostomy was performed between the terminal ileum and the transverse colon. The patient made rapid progress, and was discharged from hospital on Jan. 22. When seen six months later the liver was considerably enlarged and there were definite signs of ascites.

SUMMARY.

1. Carcinoma of the cæcum is a disease which is preponderant in the male, and occurs in the fifth and sixth decades ; the majority of cases being in the fifties.

2. The primary neoplasm is usually situated opposite the ileocæcal valve. From this fact it may be inferred that trauma is a definite factor and that the trauma is biochemical in nature.

3. The growth is invariably an adenocarcinoma. in which colloid degeneration is quite common.

4. Metastases do not occur until the symptoms have been present for many months ; consequently early removal will procure complete immunity.

5. As far as the differential diagnosis is concerned. appendicitis is suspect in the vast majority of cases. In elderly patients the presence of a tumour in the right iliac fossa and absence of vomiting should place the surgeon on his guard. A high leucocyte count is no guide. as septic absorption from the tumour in a patient with good resistance produces leucocytosis.

6. Surgical treatment should consist of : (a) An exploratory laparotomy. when ileocolostomy is to be performed should carcinoma be discovered. After an interval of from a week to ten days, this is to be followed by (b) Excision of 8 in. of the terminal ileum. cæcum. ascending colon. and hepatic flexure.

7. Where symptoms have been present for any period short of one year, anastomosis and resection offer definite cure. With a few exceptions, when symptoms have lasted more than one year. operation offers no more than one year of life. It may be fairly stated that, after the milestone of one year's symptoms is passed, the duration of the patient's life is in inverse proportion to the subsequent months.

In conclusion we should like to express our thanks to Dr. H. A. Lucas for much help in the preparation and examination of the microscopical structure of the majority of our cases. We wish also to tender our grateful thanks to Miss Mary Barclay-Smith. Mr. Ford, and Mr. Sewell for their excellent illustrations.

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A REPORT ON THE STRANGEWAYS COLLECTION OF RHEUMATOID JOINTS IN THE MUSEUM OF THE ROYAL COLLEGE OF SURGEONS.

By R. LAWFORD KNAGGS,

WITH PHOTO-MICROGRAPHS BY G. H. RODMAN.

SUMMARY OF CONTENTS.

PART I.

PART I.

HISTORY OF THE STRANGEWAYS COLLECTION.

THE Strangeways Collection of Rheumatoid Joints is one result of an effort on the part of a considerable number of medical men and their friends to provide opportunity for the careful and concentrated investigation of particular diseases which might from time to time be selected. The committee included Sir Clifford Albutt, Sir William Church, and Professor Sims Woodhead, and the first subject chosen for study was osteo-arthritis and the chronic rheumatoid conditions. Dr. S. T. P. Strangeways, who had been associated with Professor Kanthack at St. Bartholomew's Hospital, and afterwards at Cambridge, first as a Demonstrator and then as a University Lecturer on Pathology, was entrusted with the management of the inquiry (1905). He received no salary, and, moreover, the committee were indebted to him for much personal effort in collecting funds to finance the small hospital that was started. Cases and specimens were sent to him from many medical men in different parts of the country who were interested in the work, and he was able, by keeping in touch with the physicians in charge of the Poor Law Infirmaries, to obtain a knowledge of many cases, and in due course to acquire many valuable specimens.

In 1908 a special Research Hospital was erected in Hills Road, Cambridge, and here the various researches into the nature of chronic arthritis were carried out by Dr. Strangeways and those willing co-workers who from time to time became associated with him. These researches were published in the Committee's Annual Bulletins.* In the meantime the collection of pathological specimens had been gradually developing into a small museum. The work was interrupted by the War, and the taking over of the Hospital by the military

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* Dr. Strangeways contributed the following articles to the Bulletins of the Committee for the Study of Special Diseases :—

1905. " A Résumé of Some of the More Important Literature on the Etiology and Symptoms of Rheumatoid Arthritis "—with EVA McCOLL, M.D.

1907. " A Report on Some Points in the Etiology and Onset of 200 Cases of So-called Rheumatoid Arthritis "—with J. BARNES BURT, M.D.

1907. " A Study of Joints from Cases of Rheumatoid Arthritis and Chronic Gout by Means of Skiagrams and Dissection of the Affected Parts ".

1907. " An Enquiry into the Value of the Opsonic Index "—with M. P. FITZGERALD and R. J. WHITEMAN.

1907. " A Study of Skiagrams from the Hands of 100 Cases of So-called Rheumatoid Arthritis and Chronic Gout "—with J. BARNES BURT, M.D.

1908. " Conjunctival Reaction to Tuberculin in Arthritic Diseases ".

1908. " Report on a Case of Hypertrophic Pulmonary Osteo-arthropathy "—with C. PONDER.

1910. " A Study of Joints from Cases of Rheumatoid Arthritis and Chronic Gout by Means of Skiagrams and Dissections of the Affected Parts "—with EMILY H. MORRIS.

1914. " A Case of So-called Rheumatoid Arthritis due to Pneumococcal Infection ".

Proc. Roy. Soc. Med., 1923, xvii, 1.—In a discussion on the etiology and treatment of osteo- and rheumatoid arthritis, Dr. Strangeways made a valuable contribution to the study of rheumatoid arthritis. By the aid of pathological specimens he demonstrated six types of arthritis at present included under that name—viz. : (1) A capsular type ; (2) A dry type ; (3) The adhesive type ; (4) The atrophic form ; (5) The villous type ; and (6) The infective type. For further details, see the report of the discussion in the *Proceedings.*

authorities. When Dr. Strangeways was able to resume his labours he was gradually led to the study of tissue culture in its bearing upon the growth of articular structures—especially cartilage. The war, therefore, may be considered to have marked the end of the formation of the Strangeways Collection.

Dr. Strangeways died in his sixtieth year, on Dec. 23, 1926, and the Collection was offered by the trustees, Sir Humphry Rolleston, Professor H. R. Dean, and Dr. Malcolm Donaldson, to the Council of the Royal College of Surgeons, by whom it was accepted on July 14, 1927. When the Council of the Royal College of Surgeons took over the Collection, it entrusted to me the task of examining, preparing, and describing the specimens. This report contains the observations I made, and the conclusions I came to, regarding the nature of osteo-arthritis and the chronic rheumatoid conditions during the three years devoted to the investigation of the Collection.

The Collection comprises some 250 specimens of chronic arthritis and a large number of microscopical sections. There is also a typed manuscript in which Dr. Strangeways has described the macro- and microscopical peculiarities met with in his study of diseased joints. The manuscript is illustrated by photographs of specimens and microscopical sections.* These proved of great use in confirming the identity of many of the specimens and of the slides obtained from them.

The majority of the diseased joints were obtained from infirmary inmates, who in many instances were advanced in years and hopelessly crippled. Others were from young people, and many of these had been acquired after surgical excision. Most of the specimens were the subject of advanced disease ; many cases were represented by more than one joint, and in several instances a series of joints had been obtained from a single individual.

Clinical notes were supplied with a large number of the specimens ; and since the Collection came into possession of the College a good deal of valuable information has been obtained about others. Specimens of osteo-arthritis, rheumatoid arthritis, and gout formed the bulk of the Collection, but there was also an important group of Charcot's joints. Some examples of tuberculous disease and acute septic arthritis had probably been included for the sake of comparison.

THE CORRELATION OF OSTEO-ARTHRITIS, RHEUMATOID ARTHRITIS, AND GOUT.

A careful examination of the different portions of such a comprehensive collection produced a considerable modification of my previous views as to the nature of the rheumatoid affections. At the very outset two difficulties

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* Had Dr. Strangeways lived to complete his work, it is certain that he would have paid a tribute to the co-operation of Mrs. Strangeways—especially in the photographic part of it. It will be seen from the text that the photographs in the manuscript have contributed materially to placing the collection of specimens upon a satisfactory permanent basis ; but they also constitute a very useful part of the gift to the College; for the manuscript, which forms three considerable volumes, contains many excellent representations of microscopical preparations which can be consulted when the specimens are being examined. The catalogue description of each specimen illustrated in the manuscript contains a reference to the figures connected with it.

forced themselves to the front : (1) How is osteo-arthritis to be distinguished from rheumatoid arthritis, and what is the difference between them ? and (2) What is the position of gout in relation to them ?

Before these questions are considered, an epitome of the criteria generally relied upon to discriminate osteo-arthritis from rheumatoid arthritis may be interpolated with advantage.

The salient pathological features of *osteo-arthritis* are :—

1. Vertical fibrillation and splitting of the articular cartilage, which becomes worn away at the sites of pressure and friction.

2. Sclerosis, eburnation, and grooving of the underlying articular bone.

3. Osteophytic outgrowths, more particularly at the articular margins (lipping), and, not infrequently, on the articular surfaces.

Those of *rheumatoid arthritis* are :—

1. Erosion of cartilage independent of friction or pressure, e.g., at the articular periphery.

2. The formation of fibrous adhesions, and of fibrous tissue between the articular surfaces, tending to result in

3. Ankylosis, fibrous or bony.

4. Absence of lipping and other bony changes distinctive of osteo-arthritis—such as eburnation and grooving.

5. Atrophic changes, which are usually pronounced.

The clinical distinction between the two conditions is equally clear-cut. In *osteo-arthritis* there is :—

1. Pain, stiffness, and effusion into the joint, all of which may be but little marked.

2. Grating, or crepitation on movement.

3. Deformity from osteophytic outgrowths.

4. When movement is restricted to the point of fixation, it is due to locking of marginal osteophytes. It is rare to come across any adhesion or ankylosis between articular surfaces, except in certain small joints, in which, normally, movement is of a very limited character.

In *rheumatoid arthritis* :—

1. There is more evidence of inflammation—usually of a subacute character.

2. The swollen joints are apt to assume a fusiform shape owing to the involvement of the peri-articular tissues.

3. There is much pain and limitation of movement, often associated with contractures and muscular atrophy, and occasionally complicated by displacements.

4. Finally, there is a very marked tendency towards rigid ankylosis, which is sometimes bony, but more often fibrous.

THE DIFFERENCE BETWEEN OSTEO- AND RHEUMATOID ARTHRITIS.
THE TWO CONDITIONS MERGE INTO EACH OTHER PATHOLOGICALLY AND CLINICALLY.

As the investigation proceeded it soon became evident that there is no sharp dividing line between osteo-arthritis and rheumatoid arthritis. It is well known that the two conditions may merge or overlap to some extent, but I was certainly unprepared for the frequency with which, in a collection of unselected cases, some of the signs characteristic of osteo-arthritis were associated with others peculiar to rheumatoid arthritis.

Marginal lipping—looked upon as almost diagnostic of osteo-arthritis—was present in many specimens of unquestionable rheumatoid arthritis : though slight in some, it was so pronounced in others that they might easily

have been mistaken for osteo-arthritic joints, if the evidence in favour of rheumatoid arthritis had not been incontrovertible.

[Lipping arises in different ways. One form results from the collapse of very atrophic cancellous tissue underlying the articular surface, and the squeezing outwards of the marginal portion. This kind of lipping is more likely to be found in rheumatoid arthritis, but even in that affection the usual type, which depends on marginal osteophytes, is the prevailing one.]

Eburnation was present in several rheumatoid arthritic joints. It was not nearly so frequent as lipping, but its occurrence in joints which, to all
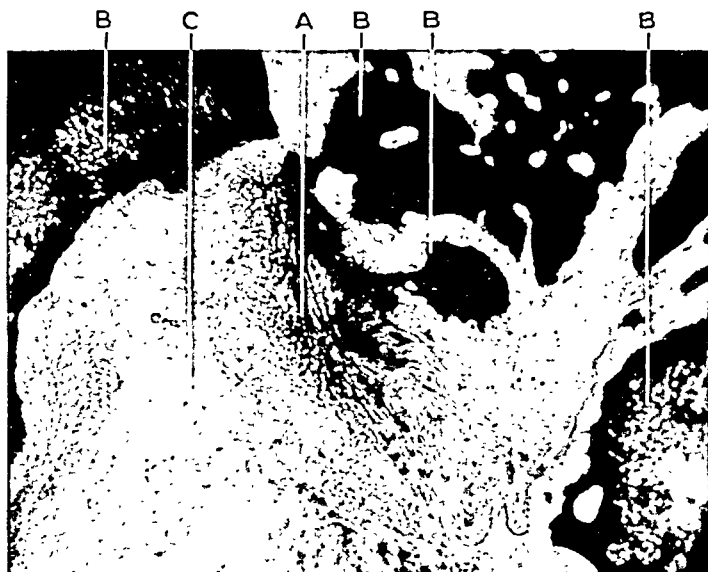
FIBROUS DEGENERATION OF BONE.

FIG. 41.—Section from the radius from a Charcot's wrist (left). Emma A., age ?. A, Trabecula undergoing resolution into fibrous tissue. B, Bone degenerating; an early stage of fibrous resolution. C, Space filled with fibrous tissue; hardly any cells present—the consequence of fibrous resolution of bone. (*Specimen* S.C. 93.) (× 50.)

appearances, had been long ankylosed from adhesions and contracted ligaments at once attracted attention. The presence of *grooving* in some instances showed that friction had undoubtedly been present in an earlier stage of the disease.

Ankylosis, on the other hand, which is an end-condition of rheumatoid arthritis, seems specially to affect certain joints in osteo-arthritis. It occurs in the vertebral, carpal, and tarsal articulations, and is doubtless favoured by the slight mobility permitted by the anatomical configuration of the joint surfaces, and by the pressures to which they are subjected. Bony ankylosis is so common in these joints that I had come to look upon it as a natural osteo-arthritic phenomenon.

Ankylosis in rheumatoid arthritis shows no such discrimination. It affects all kinds of joints—both large and small. Intra-articular adhesions,

contracted capsules and ligaments, and muscular spasm and contracture, all play their part, and the bones may be fixed together by bone or fibrous tissue.

The ankylosis in both affections is almost certainly inflammatory in origin, and must not be confused with the 'locking' which occurs occasionally in some of the large joints in osteo-arthritis—notably in the hip. In such cases the articular surfaces are usually sclerosed and eburnated, and, though in contact, are not adherent to one another.

The Difficulty of Distinguishing between Osteo-arthritic and Rheumatoid Lesions.—The macroscopic phenomena just mentioned do not exhaust the evidence of this tendency of the two forms of arthritis to merge, but they are the most striking examples. Owing to the way in which signs usually considered to be diagnostic of osteo-arthritis strayed into specimens whose general character was that of rheumatoid arthritis, it was often difficult to decide in which category a specimen ought to be placed, and only after consideration of the clinical

FIG. 42.—Fibrous resolution of bone beneath a fibrous replacement of the articular cartilage. Section from the head of a left humerus. Rheumatoid arthritis. Jessie R., age 70. A, Intermediate portion of a trabecula resolving into fibrous tissue; B, Degenerating trabeculæ; C, Fibrous covering layers, partly resulting from fibrous change in the cartilage, and partly from fibrous degeneration of bone. (*Specimen* S.C. 35—11.) (× 35.)

history, and the obvious inflammatory nature of the affection, were some of them finally labelled 'rheumatoid arthritis'. The difficulty was not a fanciful one. On several occasions expert pathologists first formed the impression that certain specimens were osteo-arthritic, but on more careful examination recognized them as examples of rheumatoid arthritis. To one authority both knees from the same individual were submitted; the disease in one was diagnosed as osteo-arthritis, and in the other as rheumatoid arthritis. Even Dr. Strangeways in his typed manuscript has figured two joints from the same case—one as osteo- and the other as rheumatoid arthritis. (S.C. 47·2, S.C. 47·4. *Figs.* in MSS. 85 and 119.)

Toxic and Other Factors in the Production of the Arthritic Changes.—

FIG. 43.—Fibrous degeneration of bone. Section from a case of rheumatoid arthritis. Sarah A. H., age 76. Left knee. A, Bone and cartilage degenerating into fibrous tissue; B, Trabeculæ fading into fibrous tissue; C, Strand of fibrous tissue joining two trabeculæ, and representing a resolved portion of bone. (*Specimen* S.C. 41—5.) (× 35.)

The bone and joint changes of osteo- and rheumatoid arthritis are thought by some to be the result of a deficiency of some diet factor, and that the two

affections are 'deficiency diseases'. The more general belief is that they are caused by toxins. I am in sympathy with this latter opinion. The toxins may be of micro-organic origin and may obtain entrance to the circulation from some distant focus of infection, or they may be formed locally in an infected joint. They may also enter the circulation by the process of 'sub-infection' described by Adami (*see* Note, p. 160, *Inflammatory and Toxic Diseases of Bone*) in Lane's *Operative Treatment of Chronic Intestinal Obstruction* (4th ed.), and others may be the consequence of disturbed metabolism (Adami, *General Pathology*, i, 342).

Whatever their source or their precise nature, it may be presumed that the toxins circulated to the different joints of the same individual would be identical, and that the phenomena produced would be of similar type, though not necessarily the same in degree. If the type differed, the presence of some other factor would seem probable.

I would suggest that a very natural factor is to be found in the vitality of the joint tissues and their ability to resist or modify the reaction that toxic damage may excite. Most people will agree that variations in these qualities exert an important influence in modifying disease; and a clearer understanding of some of the problems in the pathology of osteo- and rheu-matoid arthritis may be reached if it is assumed that such differences *do* affect the joint tissues of those who suffer from these disorders.

It is well known that the subjects of osteo-arthritis are usually robust people who have led active and even arduous lives; and that the signs, as a rule, make their appearance in middle age. Rheumatoid arthritis, on the other hand, is more apt to attack the delicate and feeble, whose health has, in many cases, been further impaired by associated circumstances; con-sequently many young people are included among its victims.

As a probable inference from these

Fig. 44.—Slight signs of fibrous resolution of bone ; early signs of cartilage degeneration. Section from a case of rheumatoid arthritis ending in bony ankylosis. Mary G., age 54. From the head of the humerus. The site of this section is more central than those shown in *Figs.* 45 and 46 from the same case, which are near the margin of the articular surface. The cartilage shows proliferation of cells and atrophy of the matrix around. A, Traces of fibrous resolution at the extremities of a tra-becula. (*Specimen* S.C. 27—10.)

facts and theories I have come to look upon the various conditions classified under osteo- and rheumatoid arthritis as representing degrees of the same process, and these degrees as signifying stages in the failing vitality of the articular structures.* The stages start from a state which represents a joint

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* Facts would seem to justify an assumption that one joint may be more susceptible to toxic damage than another, or may show an inferior power to react to it, even in the same person. This, indeed, is generally accepted, for it is recognized that a joint may be so

in full health, and descend step by step towards complete disorganization. Thus, on the step below the healthy joint comes osteo-arthritis, and the steps below this correspond to the different varieties of rheumatoid arthritis.

In osteo-arthritis the joint tissues react in a certain way to conditions—presumably of a toxic nature—that injure them, and though not irritated to the point of diapedesis, they are, nevertheless, stimulated to produce bone. In rheumatoid arthritis, on the other hand, the inflammation set up is usually accompanied by leucocytic exudation, and is frequently combined with degenerative changes in capsule, cartilage, and bone—in other words, the inflammatory response to the toxic damage is marked, but the tissues are unable to return to health, and tend to degenerate.

It will be shown that joints take on the rheumatoid arthritic character by reason of inflammatory degeneration of the articular cartilage and bone, and the capsule is also involved. This degeneration is evidence of a diminished tissue vitality; and the frequent presence of osteo-arthritic phenomena, side by side with degenerative changes, points to a period of the disorder when the tissues reacted in a healthy manner.

Final Conclusion.—The investigation of these intermediate cases in the Strangeways Collection has gradually led me to the belief that osteo-arthritis and rheumatoid arthritis are expressions—at opposite ends of the same scale —of a single disease, which I have reason to think is toxic in its origin. On this supposition osteo-arthritis and rheumatoid arthritis are not separate affections, but simply names by which two groups of characteristic pathological changes are distinguished.

When the tissues are healthy and robust the toxic irritation acts as a stimulus to growth processes, and osteo-arthritis results. When the vitality of the tissues is poor, definite inflammatory reaction may be excited, and in the event of the tissues being unable to maintain their vitality in the face of such reaction, degeneration or even disintegration results. In either case rheumatoid arthritis develops.

THE RELATION OF GOUT TO OSTEO- AND·RHEUMATOID ARTHRITIS.

Gout is very closely akin to the rheumatoid affections. A urate of soda deposit is the one diagnostic feature of gout, and in its absence it is impossible to distinguish the gouty joint from one that has the characteristics either of osteo-arthritis or of rheumatoid arthritis. Numerous specimens are included in the Collection whose gouty nature is attested by the deposit of urates. Nearly all of them show other arthritic changes, which in many instances completely overshadow the one that justifies their inclusion in the gouty category.

At the present time uric acid and the urates are believed to be a consequence of faulty purin metabolism. They are not thought to be the cause of gout, but to indicate the occurrence of certain metabolic changes, which, in

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impaired by injury, cold, or exposure as to become less able to resist infection by micro-organisms. Moreover, one case will be quoted from which it may be inferred that tissue resistance to toxic influence may vary in different parts of the same joint (*Specimen* S.C. 35–13) (*see Figs.* 49–52).

addition to the uratic deposit. " produce simultaneously other bodies having toxic effects ".   (Adami, loc. cit., i, 342.)

It may well be that, while the biurate of soda is being deposited in the joint and the surrounding tissues, the toxic bodies just mentioned set up changes in the articular bone and cartilage which are indistinguishable from those of osteo- or rheumatoid arthritis.   It is possible that all the phenomena of gout may be contained within the compass of a single pathological sequence.   At the same time it must not be forgotten that almost everyone may become the producer of such toxins as are believed to be instrumental in causing the rheumatoid affections ; and therefore the arthritic changes in the gouty joint —apart from the deposition of urate of soda and the inflammation for which it is responsible—may well be due to a twofold toxic attack ; one by toxins resulting from disordered purin metabolism, and the other by the same kind of toxins that are supposed to be responsible for the arthritic changes in osteo- and rheumatoid arthritis.

Articular cartilage and other joint tissues seem to have a special attraction for urates, and their deposition is probably the first step in that deterioration which renders a joint susceptible to toxic damage.

FIBROUS AND BONY ANKYLOSIS IN RHEUMATOID ARTHRITIS. THE HISTOLOGICAL CHANGES COMPARED.

The Strangeways Collection contains two series of specimens which I wish to contrast.

Case 1.—The first series (Group S.C. 27) was obtained from a well-nourished woman, aged 54.   The disease began in the thumb and then in the great toe when she was 18.   It progressed gradually and all the time was attended by great pain. Twelve years before death she could sit up in bed, propped up by pillows.   Then she had a bad attack of pain and never sat up again.   Her jaw was almost fixed. She could sew and feed herself almost up to the time she died.

Post-mortem.—There was advanced atheroma of the aorta, the kidneys were slightly granular, the muscles fatty, and the spine curved laterally.

Six of the ten specimens from this patient *show bony ankylosis*—namely, both knees, in which all three bones are fused, with subluxation in the left ; the left hip, which is dislocated ; the left ankle, tarsus, and metatarsus ; the right elbow and right wrist.   The post-mortem notes state that the left wrist and right ankle were also ankylosed.   The bones were very rarefied, and several fractures close to the ankylosed joints were no doubt caused by the tests for mobility at the post-mortem.

Case 2.—The second series (Group S.C. 35) was acquired from a small thin woman, aged 70.   The clinical notes tell us only that the limbs were flexed, the joint movements greatly limited, and some of the phalangeal joints ankylosed.   One kidney was granular, and pulmonary adhesions, emphysema, and pneumonia of the left base were present.

This series also contains ten specimens—namely, both hips, both knees, and a right ankle with part of the tarsus ; also both shoulders (in one the head of the humerus only) ; both elbows, and the right wrist and hand.   The bones entering into the different joints are closely bound together by fibrosed capsules, and the joint cavities are much diminished.   Capsular contraction is marked, all the 'slack' in such joints as the shoulder, elbow, hip, knee, and ankle being taken up ; for example, in the hip the capsule is attached round the margin of the head of the femur and to the edge of the acetabulum.   Quite apart from adhesions, joint movements must have been practically abolished.   But though the articular surfaces are thus held in

close contact, and were probably subjected to some degree of pressure under conditions which might be considered favourable to bony fusion, yet *no bony ankylosis is present in any of the large joints.*   Indeed, cartilage still covers the whole or the chief part of the articular surfaces, though in places it is atrophied or hypertrophied or transformed into fibrous tissue ; and whilst in some instances the joint cavities are seen between the cartilages, in others they are partially occluded by fibrous adhesions. Fusion of two bones occurs only in one of the phalangeal joints, in the carpus, and in one of the astragalo-calcanean joints.

What is the explanation of this striking difference between the two series of specimens ?   Why is bony ankylosis the rule in the first, and its absence such a noticeable feature of the second ?
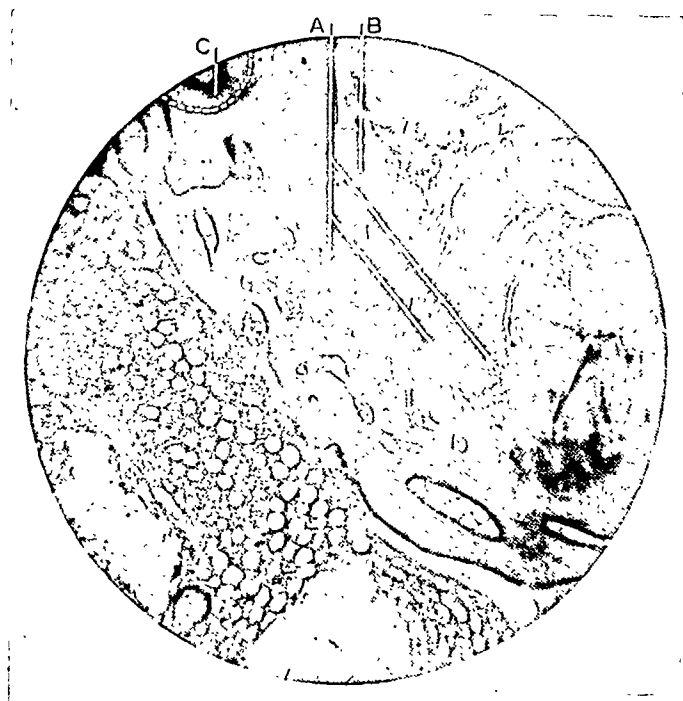
Fig. 45.—Changes in articular cartilage from a case of ankylosing (osseous) rheumatoid arthritis. Section from the head of the same humerus as *Fig.* 44, but nearer the margin. Calcification and ossification are advancing into degenerating cartilage, which is being changed into fibrocartilage on the left, and breaking up into villous processes on the right. Vascular and cellular connective tissue fills many of the spaces in the subchondroid bone and calcified layers, and medullary and fat cells occupy the spaces below.   A, Proliferating cells in spaces ; B, Fibrosing cartilage : C, Well-marked fibrocartilage.   (*Specimen S.C.* 27—10.) (× 90.)

Bony ankylosis, as a rule, begins with the fusion of granulation tissue springing from two opposing articular surfaces denuded of cartilage ; often an intrusion of similar tissue from the synovial membrane is incorporated with this interosseous bond.   Into this area of fibrosing granulation tissue trabeculæ advance from opposite sides, and coalesce ; medullary spaces form, and ultimate fusion of the cancellous tissue and medulla of the separate bones follows.   Bone is usually the result of metaplasia of the fibrous tissue.

The mode of union in Case 1 differs from that outlined above. In that case the cartilage assumes the rôle played by the granulation tissue in the typical form.

Microscopical sections were taken from a shoulder and an elbow—from a humerus in each case. In these joints no bony union had so far occurred. The chief changes are in the articular cartilage, and are very similar in both joints. The outstanding feature is the activity of the cartilage cells. The matrix shows a tendency to become fibrous, and to degenerate around the capsules. The cells within the capsules increase rapidly in number, especially
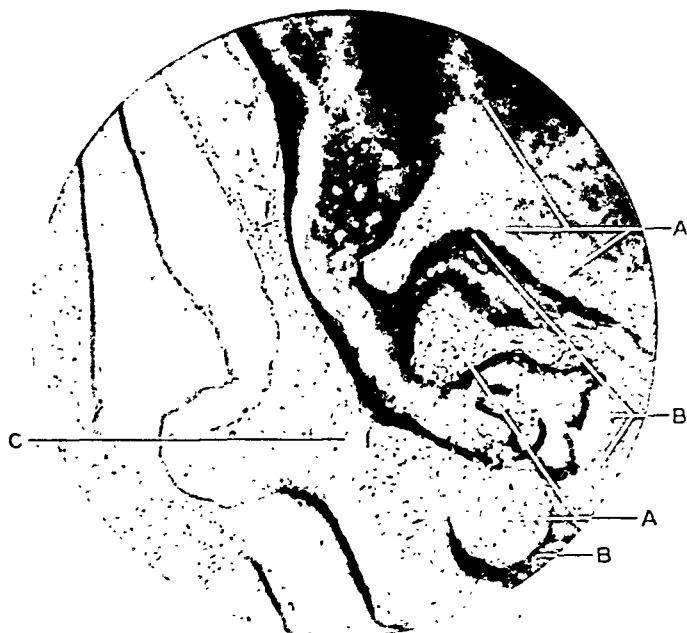
Fig. 46.—Changes in articular cartilage in a case of ankylosing (osseous) rheumatoid arthritis. From the same section as *Fig.* 45, which it joins. The crescent at the right-hand bottom corner can be recognized in *Fig.* 45 (C). A, Fibrocartilage ; B, Fibrocartilage metaplasing into bone ; C, Very vascular proliferating connective tissue. (*Specimen* S.C. 27—10.) (× 90.)

towards the surface ; they maintain a healthy appearance and stain well (*Fig.* 45). A more advanced condition shows the cartilage transformed into a very cellular fibrocartilage in which masses of crowded cartilage cells with tracts of hyaline matrix occur. The calcified zone is less regular, increased in depth, and ill-defined. Ossification is active, bone being produced by metaplasia in calcified cartilage, in fibrocartilage, and in the crowded masses of cartilage cells (*Fig.* 46). In one section a mass of fibrocartilage occupies a medullary space below a much changed portion of the articular cartilage from which it is obviously an outgrowth (*Fig.* 47). The presence of inflammatory cells in the capsule and synovial membrane put the inflammatory nature of the

joint changes beyond doubt.* Associated with these changes there are, in places, others suggestive of commencing degeneration (*Fig. 48*).

From the energetic character of the abnormal ossification going on in the articular cartilage, it may be inferred that bony ankylosis would probably result, if degenerative changes within the cartilage did not progress and prevent it.

The histology of Case 2 is illuminating. Sections from several specimens show that both cartilage and bone behave in a very different fashion to that seen in *Case* 1. In the cartilage the cells are less evenly distributed and tend to disappear, so that the fibrous transformation of the superficial layers shows only sparse elongated nuclei, whereas the deeper layers assume in some parts a fibrocartilaginous, and in others a fibro-connective-tissue character. In the

FIG. 47.—Changes in the articular surface layers in a case of ankylosing (osseous) rheumatoid arthritis. Elbow. Section from lower end of humerus. The parts shown are close to those in *Fig.* 48. A mass of fibrocartilage has formed in a medullary space and degenerative changes are going on in cartilage and bone. A, Degenerative spaces in the subchondroid bone containing fibrocellular connective tissue and giant cells; B, The mass of fibrocartilage in the medullary spaces — ossification is apparently beginning at one point by metaplasia of the fibrous tissue: C, Developing fibrocartilage. (*Specimen* S.C. 27—9.) (× 35.)

subarticular bone there is an increase in the size and apparently in the number of the bone-cell cavities, and traces of fibrous tissue can often be seen in the substance of the trabeculæ (*Fig.* 42). From the margins of the subchondral bone multitudinous fibres stream outwards and blend with the fibro-cartilage and the fibro-connective-tissue, so that the transition from bone to the altered cartilage is gradual and almost imperceptible (*Fig.* 43).

FIG. 48.—Changes in the articular surface structures in a case of ankylosing (osseous) rheumatoid arthritis. Mary G., age 54. Left elbow. Section from lower end of humerus. Calcification and ossification are advancing into degenerating cartilage. The advanced margin of calcification (B) is defined on the left in the middle depth of the cartilage. Above it there is fibrocartilage to the surface, and fibres run into it. A number of small spaces (A) in the subchondroid layer of bone, and two or three involving the cartilage, contain proliferated connective tissue. (*Specimen* S.C. 27—9.)

* The sustained inflammatory reaction going on to diapedesis in *Case* 1 may be compared with the changes usually seen in osteo-arthritis. In that disorder the chief evidence of inflammation is the proliferation of the connective tissue and the stimulation of ossification, but without diapedesis, except of the most trivial character.

Medullary spaces bounded by these degenerating trabeculæ contain a varying amount of fibrous tissue with which the numerous fine fibrils emerging from the bone intermingle (*Fig.* 50). Cells, some probably liberated and altered bone-cells, are numerous in the immediate vicinity of the resolving bone. It is evident that the superficial trabeculæ are slowly disappearing, and being replaced by fibrous tissue. The fibrosing cartilaginous covering is thus being continually recruited on its deep surface, whilst the level of the trabeculæ in contact with it becomes irregular, and tends slowly to recede.

This process of resolution of bone and cartilage into fibrous tissue was present in most of the specimens from S.C. 35 microscopically examined : but in the left elbow a more advanced condition was found. The opposed surfaces of the humerus and radius were eburnated and sclerosed ; small gaps in the surface bone were filled

Fig. 49.—Rheumatoid arthritis. Fibrous ankylosis. Left elbow-joint. Antero-posterior section through humero-ulnar portion. Jessie R., age 70. Fibrous adhesions are present between the articular surfaces ; also rarefaction of the cancellous tissue and sclerosis in the vicinity of the joint. Fibrous degeneration of bone is especially marked in the sclerotic parts. (*Specimen* S.C. 35—13.) (× 2·5.)

with granular-looking fibrous tissue (resolved portions of bone) which preserved the smooth unbroken level. The trabeculæ for some depth were composed of massed calcareous granules, stained purple by hæmatoxylin, and held together by a fine fibrous groundwork. Some of these degenerating trabeculæ still contained portions of bone having a blurred and granular appearance. Irregular lines and fissures were numerous in them, and evidently some of the trabeculæ were ready to break up into fragments and granular débris such as could be seen strewn in some places in the soft tissue. At one part on the surface of one of the bones was a long tract of crowded calcareous particles beneath a thin layer of degenerate cartilage, which was broken up into a fringe of short villi. There was no cohesion of the calcareous granules, and the tract had evidently resulted from disintegration of the calcified zone. In the sclerosed bone beneath it the tra-

Fig. 50.—Fibrous degeneration of bone. Rheumatoid arthritis. A more highly magnified part of *Fig.* 49. The upper edge of the photograph adjoins the articular cartilage. The sclerosed subchondral bone undergoing fibrous degeneration. A, Points where fibrous resolution is well seen ; B, Small spaces formed in the bone. Contents : fibrous tissue, connective-tissue cells, new vessels, a little fat, and some giant cells. The larger spaces are probably formed by the confluence of smaller ones. (*Specimen* S.C. 35—13.) (× 35.)

beculæ presented the peculiarities just described. (*Fig.* 51.)

At another part of the section a striking appearance was produced by

several trabeculæ, parts of which were dead and parts alive. The dead parts were partially detached, and were composed of coarse fibrous tissue enclosing a number of swollen dead bone-cells. It was evident that in some parts of this joint, tissue death had occurred, and disintegration and not resolution into fibrous tissue was taking place. (*Fig. 52.*)

Different theories might be suggested to account for the peculiar changes met with in this case. The simplest would be that the vitality of the joint tissues was of so low a grade that reaction to toxic irritation was practically impossible, and slow degeneration, as evidenced by a reversion to a less highly organized form of connective tissue, resulted. In some places the tissue vitality was probably below the general low level, and at those points necrosis occurred. The histological evidence explains the absence of bony ankylosis.
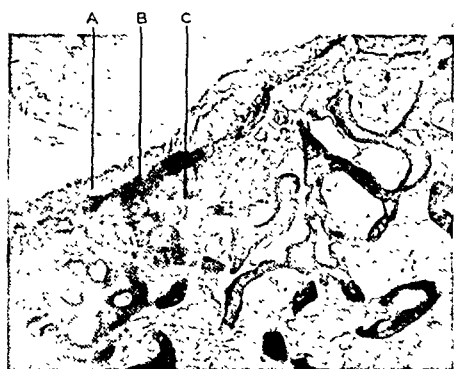
FIG. 51.—Rheumatoid arthritis. A degenerating articular surface from the same elbow as that shown in *Fig. 49.* The photograph was probably taken from the radius; the opposing bone shows very similar conditions. A, Necrotic cartilage; B, Layer of disintegrated lime granules, staining purple with hæmatoxylin—the calcified layer; C, Superficial trabeculæ undergoing granular disintegration: fibrous tissue in the medullary spaces, in which strands of calcareous granules, as well as débris, can be seen. The trabeculæ are crumbling at their edges and apparently resolving into delicate fibrous tissue, in which there are no cells. (*Specimen* S.C. 35—13.) (× 25.)
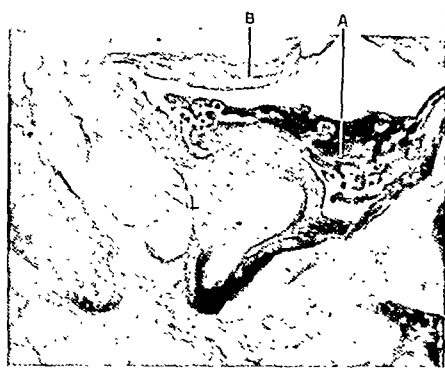
FIG. 52.—Rheumatoid arthritis ending in fibrous ankylosis. From the same section as *Fig. 51.* The radius. A, Dead swollen bone-cells in a portion of a trabecula largely separated into a space, possibly resulting around it within bone. The dead portion is largely degenerated so that only a thin infiltration of lime salts maintains its shape. B, Degenerated deep portion of the subchondroid bone layer, above which is the fibrous layer resulting from degeneration of its superficial portion, of the calcified layer, and of the articular cartilage. A fibrous ingrowth from the synovial membrane blends with it. (*Specimen* S.C. 35—13.) (× 50.)

That is obviously impossible between opposing bony surfaces undergoing necrotic disintegration or resolving into fibrous tissue, especially when, as in the latter case, the fibrous tissue between the bones is being augmented in the process.

The two cases represent two clearly defined forms of rheumatoid arthritis. Both are inflammatory, though only in the first case is the condition marked by migration of leucocytes to a notable degree; yet the absence of this unmistakable sign of inflammation in the second case is easily understood. Bony ankylosis was the usual termination in the first case, and failed in the second owing to the degenerative changes consequent on the low-grade vitality of the tissues.

A further insight into the nature of these conditions may be obtained by a study of the changes present in those cases in which the signs of osteo-arthritis and rheumatoid arthritis are combined.

Case 3.—A woman, age 76, is represented by nine specimens (Group S.C. 41) and in none of them is there bony ankylosis. The joint disease lasted twenty-five years, and its onset had been rapid. All the joints were involved in the first three weeks, and she was crippled in the course of a few months. After death a chronic empyema with a thick wall was found in the left pleura. It contained a pint of pus. Pure cultures of streptococci were obtained from the pus, and also from rabbits that died after inoculation with it. The hypertrophic manifestations were clearly of long standing. Marginal lipping was pronounced, and in the knees, where the different bones had been in contact, the articular surfaces showed flat plate-like elevations.
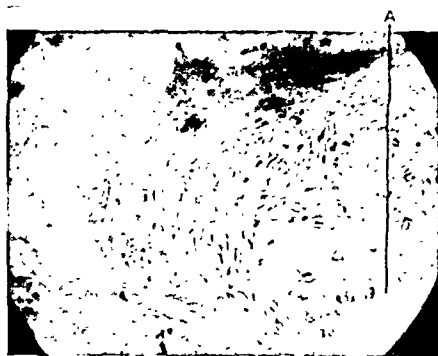
Fig. 53.—Rheumatoid arthritis. To show bone metaplasing into fibrous tissue. From the same section as *Fig.* 49. A, Bone. (*Specimen* S.C. 35—13.) (× 125.)

The microscope shows the principle seat of mischief to be where the cartilage joins the bone. The calcified zone and the subchondral layer of bone have been largely destroyed. A row of spaces is present at the line of junction, and they tend to spread along it and coalesce. The spaces have evidently resulted from the fibrous degeneration of the bone and calcified cartilage, and contain many stellate cells and vessels lying in a meshwork of fibrous tissue. By this process a considerable thick-ness of fibro-cellular tissue has come to lie between the cartilage and the cancellous bone-end, and the resolving edges of the adjacent trabeculæ and the deep fibrosing layers of the cartilage tend to blend with it. Patches of granulation tissue occur here and there within it, and many inflammatory cells are present in the medullary spaces below it. There is no sign of recent ossification, and as the articular surface is constituted by the superficial layers of the cartilage, and, where they have dis-appeared, by the fibrocellular layer, the fibrous form of ankylosis is in consequence assured.

The hypertrophic bone formations are the subject of degenerative changes equally with the rest of the articular surfaces—a circumstance that is strongly suggestive of their development before the ability to react had given place to degeneration.

A consideration of the foregoing facts points to the conclusion that it is the onset of degeneration that determines the fibrous form of ankylosis, and its essential feature is resolution of the articular bone and cartilage into fibrous tissue.

THREE CASES OF RHEUMATOID ARTHRITIS IN
YOUNG PEOPLE.

The cases that furnished the material for the foregoing observations were either of late middle age or advanced in years. The cases that follow occurred in childhood or adolescence. Two, like *Case* 1, developed bony ankylosis ; the other showed no such tendency and resembled *Cases* 2 and 3.

Case 4.—The disease began in Maud B. in a metacarpo-phalangeal joint when she was 18 years old. One hand and wrist and the feet and ankles next became involved. Both elbows and both knees were excised by Mr. Zachary Cope at the age of 30, but the left knee was not kept and there is some doubt as to the nature of the ankylosis present. Now at 37 years of age (1929) the left hip and shoulder, both wrists and ankles, various finger-joints, and the occipito-atloid and atlo-axoid joints are rigidly fixed, and bony ankylosis is probably present in some but not in all.

There was nothing definite in the history to account for the onset, but two years later some bad teeth were extracted, and an abscess of the breast was treated. There was a slightly rheumatic family history. The question of gonorrhœa was definitely eliminated in hospital. Amenorrhœa lasted from the twentieth to the twenty-seventh year, when a leucorrhœal discharge set in and has continued. Her periods are now regular.

The operations seem to have had a very beneficial effect on the course of the disease, which is apparently quiescent. The specimens (Group S.C. 28) include both elbows, in which all three bones are fused, and the right knee, in which the patella is ankylosed to the femur by bone.

Case 5.—Alice R, age 21, was 7 years old when her hands, wrists, and ankles, and then her knees, became swollen and painful. At the age of 9 they were 'set fast'. At 21 practically every joint in her body was involved, and wrists, elbows, knees, and hips were ankylosed. Movement in the small joints was limited. At 18 she had hæmoptysis. She never lost her cough. She had also a left purulent otorrhœa and offensive breath. She died at the age of 41 of tuberculous peritonitis and pneumonia, 'crippled, deformed, and phthisical'.

The specimens (S.C. 24) are two elbows showing bony ankylosis. They were excised when the patient was 21. Skiagrams show bony ankylosis of both knees, and of one superior tibiofibular joint.

No microscopical sections could be taken from these two cases.*

Case 6.—Elizabeth F. had her tonsils removed when she was 12 for frequent attacks of tonsillitis. Otherwise she was quite healthy till she was 13½ years old, when the joint affection began with pain in the right wrist and swelling of the knees. At the end of five weeks all her joints were affected, including the mandibular. She was an in-patient at several hospitals during some years, and at 17 was chair-ridden, with bent legs in which movement was very limited. At 18 she suffered from influenza and was again an in-patient at St. James's Hospital, Balham, where she remained six and a half years. When she was 21 both knees were excised by

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* The following case was under the care of my colleague, Mr. Edward Ward, of Leeds. It shows a certain similarity to *Cases* 4 and 5, but is a variation from the progressive and more usual type of rheumatoid arthritis :—

Percy B., age 13, a healthy-looking boy, suffered when 2 years old from some illness which was associated with pain in both legs. The legs gradually became flexed at a right angle, and when he was 5, were permanently stiff. Both knees and both ankles were ankylosed, but no other joints, and he had no pain. Mr. Ward removed wedges of bone from both knees, and sagittal sections disclosed intimate union between the femur and tibia in both. The patellæ were not ankylosed. (*Leeds Medical School Museum*, A 568.)

Mr. Zachary Cope, and in 1929 (age 27) her condition was much improved and the disease quiescent. She was chiefly inconvenienced then by the disabilities incidental to the much restricted movements of the different joints (shoulders, elbows, wrists, hands, hips, and ankles) which necessitated a reclining position.

The only specimen preserved is a sagittal section through the right knee in which all three bones are in close fibrous adaptation in a position of flexion (S.C. 51).

The microscopical examination shows that resolution into fibrous tissue is going on in the cartilage and bone forming the articular surfaces of all three bones. The degenerative changes are practically identical with those seen in *Case* 2, but inflammatory tissue is more in evidence.

No doubt in elderly people the tissues generally, including those of the joints, are much less robust than in youth, and ossification is naturally more active in the young. Consequently bony ankylosis may be expected to be more common in early life, and infrequent when the affection develops in middle life or old age. These juvenile cases show that the form of arthritis which a susceptible joint may develop in rheumatoid arthritis does not depend upon age alone, and the fact that in *Case* 6 the disease ran its course and reached its climax before the patient was 20 proves that fibrous degeneration of bone and cartilage is not a purely senile change.

(To be continued.)

IMPLANTATION OF THE URETERS FOR INOPERABLE VESICO-VAGINAL FISTULA AND ECTOPIA VESICÆ: A NEW TECHNIQUE.*

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THE problem of devising a simple one-stage operation of implanting the ureters into the bowel has long been occupying my mind, for if the blood-urea estimation and general condition of the patient are fair, there would seem no adequate reason for failure, provided the technique is simple and within the means of the general surgeon or pure gynæcologist.

It is of course admitted that the results of a two-stage operation are excellent. The statistics of Fraser, the pioneer of this operation, Gow, and myself in India, and those of Coffey, Grey Turner, Jocelyn Swan, Nitch, and others in the West, all amply demonstrate that fact; but it seemed to me that one should be able to obtain Coffey's results, in the one-stage operation, if a simpler and more rapid technique could be discovered; for such an operation in the hands of a quick surgeon is called for in cases of ectopia vesicæ, inoperable and inaccessible vesico-vaginal fistulæ, carcinoma of the bladder, and certain gunshot injuries of the pelvis, and occasionally in post-radium bladder fistulæ.

HISTORICAL.

Between the years 1899 and 1903, Peters in Canada and Lendon in Australia were the first to publish successful cases of transplantation of the ureters. Since then, surgeons all over the world have endeavoured to improve the technique. In the early days of the operation, surgeons transplanted the ureter-bearing area of the bladder, with its valve mechanism, into the bowel, but their results were only successful in 50 per cent of the cases, owing to the fact that the physics of the living was not appreciated—that is, they failed to differentiate and separate the function of a sphincter from that of a valve, and failed to recognize that urine is delivered, after operation, from the duct of an organ which works under low pressure into a receptacle where the pressure is higher.

It is to Robert Coffey that surgeons owe the elucidation of previous failures, for he followed out Harvey's injunction " study and seek out the secrets of Nature by way of experiment ". In 1908, when working with the Mayo brothers, investigating pancreatic surgery, he demonstrated that when the common bile-duct was transplanted by the direct method, it had become

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enormously dilated when observed a few weeks later. This accidental discovery led him to the solution of the mechanism used by Nature when secreting fluid at low pressure into a cavity of higher pressure. Carrying his investigations further, he discovered the secret, so far hidden from surgeons and anatomists alike—namely, that in the case of such a tube as the ureter the duct first penetrates the muscle, then runs immediately beneath the loose mucous membrane for a distance before it emerges into the lumen of the bowel. This being so, in order to achieve success in such a case as implantation of the ureters, it was essential to devise an operation which copied the valve mechanism found in Nature. Experimenting on dogs, Coffey's logical deductions were completely justified, and within the next few years Dr. Mayo, giving all credit to the brilliant conception of Coffey, operated with success on a number of cases of ectopia vesicæ, dealing with one ureter at a time.

In 1925 Coffey, having done many of the two-stage operations of transplantation of the ureters which he had originally planned, became dissatisfied with the misery and delay entailed, and therefore commenced a series of experiments, using ureteric catheters as a means of conveying urine from the kidneys in order to obviate the risk that he had observed of an inflammatory exudate in the gut wall compressing the ureter and so predisposing to uræmia. After many trials and ups and downs of technique he evolved the admirable operation which was published in the *Annals of Surgery*, in June 1927, for bilateral transplantation of the ureters, and in November, 1929, he published an inspiring report of 20 cases operated upon by this method with only 1 immediate surgical death.

While all honour must be rendered unto Coffey for the original research work, it must not be forgotten that Sir Harold Stiles in Edinburgh, and later Grey Turner in Newcastle, from 1911 onwards, were performing a two-stage operation of implantation of the ureters, using a technique much after the fashion of Witzel's gastrostomy ; Grey Turner recording 17 cases with 4 deaths.

The first implantation operation in the Eden Hospital, Calcutta, was performed in 1912 on a girl of 14 suffering from exstrophy of the bladder. Briefly stated, Peters' technique was followed. After a stormy convalescence complete cure resulted. The patient was seen from time to time, and in 1926, when she was 28 years of age, she came to hospital nine months pregnant. Cæsarean section was performed with complete success.[1]

The admirable results reported by Fraser, in Madras, of cases of the two-stage operation for inoperable vesico-vaginal fistulæ inspired Gow and the writer to follow his technique, and our results of this two-stage operation are commendable. But for a long time it has seemed to me necessary to devise a simple operation with all the advantages of Coffey's method without its complicated technique—a method which would eliminate the dangers and disappointment of two anæsthetics on cachectic young or old patients.

The accompanying series of diagrams (*Figs.* 54–56) will not only explain the technique used in my first series of operations for bilateral ureteric implantation, but will make clear the two-stage operation to those surgeons who have as yet not attempted it, or have no desire to run the risk of transplanting more than one ureter at a time.
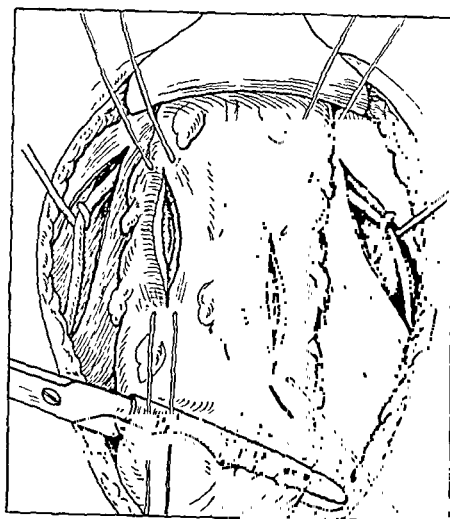
FIG. 54.—Before making the two gutters shown here on each side of the longitudinal band, the left gutter being at least 1 in. below the level of the right, long silk threads have been passed through the bowel muscle above and below the estimated incisions. Traction on these threads shown above permits the bowel wall to be held taut while the incision down to the mucous membrane through the muscle is made.

FIG. 55.—Catgut threaded at both ends with intestinal needles is passed through the lumen and wall of the ureter. The ureter is carefully laid in the gutter of the bowel and the two needles are passed through a stab wound made in the mucous membrane at the lowest portion of the gutter. The needles are made to emerge ¾ in. below the gutter and about ¼ in. apart. The two threads are then gently drawn upon until the cut end of the ureter is well through the stab incision. The catgut threads are then tied, so anchoring the ureter gently to the mucous membrane of the bowel.
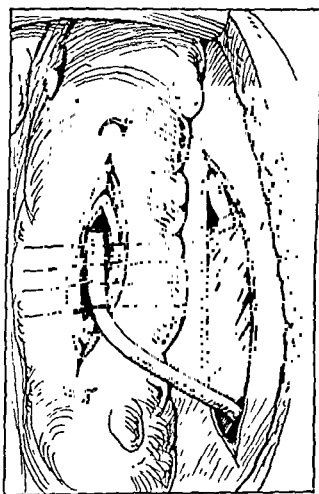
FIG. 56.—The ureter in position, four ligatures of catgut are passed from one side to the other through the peritoneum, muscle, and wall of ureter to the opposite side and then tied. The stab wound in the mucous membrane is separately sutured with three catgut sutures which pass through the ureter and the mucous membrane on each side, so preventing any back leakage from the bowel through the stab incision.

CASE REPORTS (*Group I*).

Case 1.—S., age 20, admitted for dribbling of the urine, subsequent to difficult childbirth one year ago. There is atresia of the vagina, which feels like a cartilaginous canal. The fistula is on the anterior wall, the size of one rupee. There is a history of one month's amenorrhœa. Blood-urea 0·015 per cent. General condition fair.

Both ureters were transplanted at one sitting, and except for twelve days' post-operative fever, recovery was uneventful.

At the time of operation she was seen to be pregnant. On April 3, 1931, she was re-admitted in labour and was delivered by Cæsarean section of a healthy child, leaving hospital fifteen days later.

Case 2.—H., age 18, history of still-birth and difficult labour eight months previously. The vagina admits one finger only, and in the vault of the vagina there is a fistula with a sharp fibrous margin through which the upper surface of the bladder projects. The pelvis is rachitic. General condition fair.

Both ureters were implanted on Sept. 1, 1930. The following day the general condition was good; temperature 100·5°; 23 oz. of urine had been collected from the rectum. The next day she passed 34 oz., on the third day 16 oz., and on the fourth day only a few ounces were passed. She rapidly sank into coma and died of uræmia that night. Post-mortem was refused, but there was no distension or peritonitis.
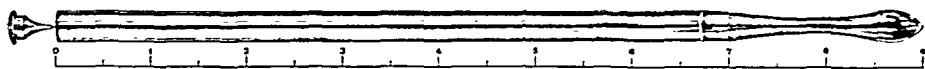
Fig. 57.—A full-size prostatic catheter from which the curved end has been removed and a screw bulb-end substituted. Total length 9 in.

I am convinced that the ureters in this case were compressed by an inflammatory exudate which dammed back the flow of urine, precipitating uræmia. For this reason I did not feel justified in further attempts to implant both ureters at one sitting until I could devise some method of getting over this difficulty of an inflammatory exudate compressing the soft ureter. During the interim therefore I went back to the two-stage operation and implanted many single ureters at a time without a death. But I was not satisfied, and determined to try again, using a means readily accessible to any surgeon—namely, the straight portion of a metal prostatic catheter, the curved part of which was cut off and a straight conical screw bulb substituted (*Fig.* 57). When all is ready the metal tube is passed through the stab incision down to the anus, and its canal is used as a tunnel to transmit the ureteric catheter to the rectum and anus without bother or kinking. (*Fig.* 58 will, I trust, make the following account intelligible.) But before proceeding I will describe the technique first used for one or both ureteric implantation operations.
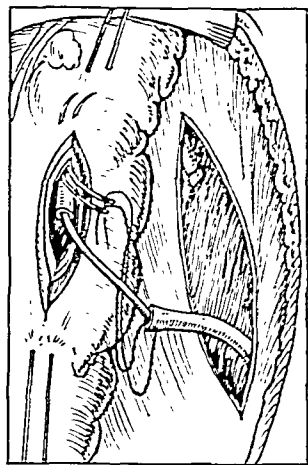
Fig. 58.—One ureter has been tied off close to the bladder and cut. As before, catgut threaded at both ends has been passed through the lumen and wall of the ureter. A ureteric catheter has been passed up the ureter for 5 or 6 in. The proximal end of the catheter is then passed down a straight silver tube, the proximal end of which will be seen emerging from the stab incision.

TECHNIQUE.

For nine days before operation the rectum is washed out daily with saline or weak permanganate solution. For the first three days the patient is given the following prescription four-hourly :—

Potas. Cit.		Glucose	℥iij
Mag. Carb. Pond. āā. gr. xxx		Aqua	℥j

The second three days 1 drachm of acid. sodii phosph. in water is given first thing in the morning, followed by 10 gr. of urotropine three times a day; the third three days the former alkaline mixture is taken.

Directly the patient is under the anæsthetic, an assistant very slowly perfuses 300 c.c. of a 20 per cent solution of glucose into the vein.

After opening the abdomen with the patient in the Trendelenburg position, the intestines are walled off in the usual way; the right ureter is quickly dissected out down to the bladder, leaving as much of its peritubular fascia around it as possible, and a piece of linen tape or a small blunt hook is passed under it. The ureter is not cut and made ready for implantation until the next stage of the operation is complete.

The sigmoid is held up by two silk threads which are inserted about 3 in. apart. A gutter is now made in the bowel wall 1½ in. long between the two silk threads, which are held taut. The handle of one's knife makes an excellent reflector of muscle and peritoneum. This stage of the operation takes only a few minutes.

The ureter is now caught up by a rubber-covered Kocher forceps, tied, and cut near the bladder, the distal end being touched with carbolic. The proximal end is brought up to the surface, and the pen-cut proximal lumen of the ureter is traversed with catgut, threaded with a needle at both ends, and a No. 12 flute-ended opaque catheter is passed for 5 or 6 in. up the ureter. The ureter is bound to the catheter by a single loop of 000 catgut.

A stab incision is made at the lower end of the gutter into the colon, and a straight short metal tube with a screw top is passed through it down to the anus. An assistant grasps it, unscrews the cap, and then the proximal end of the catheter is passed down the tube until the lie and line of the ureter are in the gutter previously made. *The metal tube is then pulled down and out of the rectum and reboiled ready for the other ureter procedure.*

In some of the early cases I had considerable difficulty in coaxing the metal tube down to the anus sufficiently far for my assistant to reach it with his fingers. I have now countered that trouble and source of delay by the simple procedure of passing into the rectum before the operation a medium-sized Kelly's cystoscope loaded with its blunt-nosed plunger, just as one would pass a proctoscope. When the stab incision referred to above is made in the bowel wall, my assistant directs the cystoscope towards the incision, the plunger is then removed and the metal tube containing the catheter is passed directly into the lumen of the cystoscope, and pushed through from above out of the proximal handled end of the cystoscope. This manœuvre is simple and quick and has given great joy since its discovery.

The double threaded catgut needles are then passed through the stab incision to emerge ¾ in. below the gutter and about ¼ in. apart. They are

drawn upon, thus bringing the ureter down through the stab wound into the bowel ; the catgut threads are then tied, thus loosely anchoring the ureter to a point at least 1 in. below the stab incision. Three or four interrupted fine catgut sutures are then passed from one edge to another, picking up the peritoneum, muscle, and ureter in order to close the incision and secure the position of the ureter. The edges of the stab incision are brought in close contact by fine catgut which traverses the wall of the ureter, thereby further anchoring the ureter and preventing back leakage from the rectum. The remaining portion of the catheter-containing ureter in the gutter is then covered by a continuous Lembert suture up to the site where the ureter emerges from its bed under the initial incision in the peritoneum as in Coffey's illustrated article.

Exactly the same technique is adopted on the left side with the left ureter except that the incision in the bowel wall is on the left of the longitudinal band, and about 1 in. below the level of the right gutter.

The two subperitoneal tunnels from which the ureters were brought up are now closed with catgut, and after a look round to see there is no oozing or any retroperitoneal hæmatoma forming—all towels are removed—the omentum is brought down and the abdominal wall is sutured in the usual way. No drainage is used. In all eight cases now to be recorded the urine began to flow at once from each catheter. In order to prevent kinking or pressure on the catheters they are threaded through a short flatus tube which is passed through the anus. The ends of the catheters are inserted into a measured quantity of carbolic solution.

CASE REPORTS (*Group II*).

Case 3.—I., age 22, history of one difficult labour. Now almost complete atresia of the vagina, the fistula being visible at the top of the canal, which admitted the little finger only. There was in addition a stone in the bladder the size of a hen's egg and a recto-vaginal fistula high up. Blood-urea 0·022 per cent, Hb. 55 per cent, R.B.C. 2½ million. On Aug. 26, 1931, the stone was removed after incising the vagina.

A fortnight later, under pernocton and ether anæsthesia, both ureters were transplanted into the rectum by the method described. The patient ran a temperature for twenty days but made an uninterrupted recovery, the recto-vaginal fistula being successfully operated upon on Nov. 13, 1931.

Case 4.—B., age 20, admitted on Sept. 4, 1931. History of one difficult labour. The whole of the base of the bladder and urethra have sloughed away, leaving a large gap surrounded by dense fibrous tissue. The patient was pregnant two months. Blood-urea 0·02 per cent, Hb. 50 per cent, R.B.C. 3 million.

Transplantation of both ureters, as above, was performed on Sept. 14. Except for slight fever for the first ten days and the fact that the patient normally aborted on the eighth day after operation, recovery was uninterrupted. She left hospital cured on Oct. 15.

Case 5.—B., age 40, admitted on Oct. 7, 1931. History of three difficult labours—the last resulting in the formation of a fistula, the size of a rupee, at the junction of the cervix and bladder, surrounded by dense scar tissue. Blood-urea 0·04 per cent, Hb. 70 per cent.

On opening the abdomen there was a large right tubo-ovarian hydrops. This was first resected. Both ureters were then transplanted as above. The patient was in excellent condition, passing urine plentifully for the first three days, but on

the fourth day she suddenly became cyanosed, coughed up blood, and died of pulmonary embolism.

Case 6.—R., age 18, admitted on Oct. 8, 1931, with a large fistula involving the urethra and bladder the result of difficult labour with dead child eight months previously. Blood-urea 0·036 per cent, Hb. 70 per cent.

Careful vaginal dissection failed to approximate the edges. Laparotomy was done on Dec. 7. Both ureters were implanted, using opaque catheters. An interesting feature of this case was the presence of two ureters on the left side lying collaterally. The upper ureter, being twice the calibre of the lower one, was the one implanted. This condition of double ureters is recorded by both Grey Turner and Thomson-Walker. Recovery after ten days' slight pyrexia was uneventful, both implanted ureters draining perfectly. There was just the scantiest wetting of the bedding from the vagina, owing to the lower ureter slightly functioning. It is probable that this extra ureter will cease to function; if not, it will be implanted separately later. The patient is walking about and quite happy on the fifteenth day after operation. On Feb. 17, 1932, the extra ureter was dealt with. She left hospital cured on March 1.

Case 7.—F., age 18, admitted on Jan. 11, 1932, with an enormous rigid hole at the base of the bladder due to difficult labour one year ago ; the baby was born dead. Hb. 45 per cent, blood-urea 0·025 per cent. Transplantation of ureters on Jan. 25. Urine drained well from the start. Had double rise type of fever for three weeks which reacted to urea-stibamine and was shown to be due to kala-azar. She left hospital happy on March 5.

Case 8.—A., age 19, admitted on Feb. 2, 1932, with an atresic vagina and unapproachable fistula due to labour eight months previously. Hb. 50 per cent, blood-urea 0·63 per cent, R.B.C. 2½ million. Under pernocton and ether both ureters were transplanted on Feb. 15. Recovery uneventful. Discharged cured on March 11.

Case 9.—S., age 30, admitted on Feb. 19, 1932. Her condition was the same as the previous case, and has existed since her last child was born dead twelve years ago. Hb. 46 per cent, blood-urea 0·03 per cent. Operation as before, but on the third day the temperature went up to 105° and remained high despite all treatment. She died of uræmia on March 19. Post-mortem refused.

Case 10.—A., age 28, admitted on March 12, 1932, for a fistula admitting two fingers at the top of a lacerated vagina. She had had twelve labours, all at full term, and all the babies had been born dead. The fistula had only occurred after the last childbirth. Both ureters were implanted on March 21. For the first eight days all went well, but the patient then grew very boisterous. The temperature became subnormal. She passed only 12 oz. of urine for the next two days, and then sank into coma, dying on March 31. At autopsy, both kidneys were large, sacculated, and full of pus, from which *B. coli* and staphylococci were cultured.

OPERATION COMMENTS.

Some surgeons insert a suprapubic drain for twenty-four hours. The experience gained from a large number of both classes of operations convinces me that this is not necessary, all the patients having healed by first intention. Grey Turner[2] implants both ureters on the right of the colon and into the longitudinal muscular band, one 2 in. above the other, the left ureter being passed under the colon. I think this must be a more difficult procedure and conducive to oozing.

In some cases the ureter is greatly dilated owing to obstruction by old inflammatory tissue in the parametrium. When inserting the catheter in these cases it is wise to run a purse string round the end of the ureter so as to

grasp the catheter lightly.   In order to secure good vision in the field of operation a large Doyen retractor is better than anything else.   Before closing the abdomen the uterus should be ventro-suspended to prevent adhesion of its posterior surface to the bowel and subsequent pelvic disorders.   All patients should be warned in the event of pregnancy to attend an antenatal clinic and come to hospital in their eighth month for Cæsarean section at term (*see Case* 1).

All patients, whether the technique be that of a one- or two-stage operation, run a temperature for ten or twelve days afterwards, presumably owing to mild pyelitis.   The first case in 1912 of ectopia vesicæ, with Cæsarean section in 1926, illustrates the small chance of permanent injury to the kidney, if that organ is healthy at the time of operation.   All patients are given copious alkaline drinks and fruit juice after operation.   In both operations castor oil is given on the fourth morning.

In the two-stage operation, where no catheter is used, the rectal tube is removed about the fifth day.   In the one-stage operation, using ureteric catheters, they are drawn out on the fifteenth day if they have not come out before.   Coffey tightly binds the distal half-inch of his ureters to the catheters in order to prevent intestinal infection reaching the ureter, but such a procedure, I think, must make difficult the removal of the catheters.   Moreover, by causing sloughing of the ligated portion of the ureter it would seem to me to risk defeat of the very object for which the catheters are tied in.

In one of my two-stage operations there was quite severe arterial bleeding, a few hours later, from the rectum, no doubt owing to my having cut a branch of the hæmorrhoidal artery.   The hæmorrhage was checked by iced water enemata containing adrenalin.

In future cases I intend to follow Wilkie of Edinburgh's custom, and give nucleic acid and *B. coli* vaccine to all patients prior to operation.   Moreover, I am of the opinion that patients in the Tropics, anæmic and debilitated, over the age of 25, will not stand the strain on both kidneys that double implantation demands, and therefore I shall in such persons use the same rapid easy technique on two separate occasions.

Ureteric catheters are not expensive, but it is important that they should be new, and at least No. 12 to No. 18 in size, with flute ends.   The use of catheters eliminates the dangers of inflammatory exudate or peristalsis compressing the soft non-tensile ureter during its course through the gutter valve in the bowel wall.

A great many of our fistulæ patients are cases with small round pelvis or osteomalacia, in whom the great damage to the cervix, bladder, and vagina in the course of long unaided labours in distant villages is easily explained.   But despite the fever, sloughing, and atresia that occur it is remarkable to observe in practically all, that on opening the abdomen, the uterus is mobile and the Fallopian tubes are normal and patent.   Again, it is interesting to note that in two cases (*Cases* 1 and 4) both patients had become pregnant just before operation despite large high vesico-vaginal fistulæ.   In this connection it may be of interest to note that on April 16, 1932, a Mrs. C. was admitted for confinement who had been born in this hospital eighteen years previously with an imperforate anus and congenital high rectovaginal fistula.   Cæsarean section was performed, and she left the hospital with her infant on May 5.

Clinically it has been a pleasure to see the joy and growth in weight of these patients as they come to realize that the misery of their previous condition has been or will be alleviated. I have found that after two or three weeks the anal sphincter becomes so alert that they can retain quite a surprising quantity of urine in the descending colon for three to four and a half hours by day and at least six hours at night.

It is admitted that these eight case reports of successful implantation of both ureters, using opaque catheters and a metal tube to transmit the catheters to the anus, is but a small number from which to draw conclusions. But it is hoped that surgeons who have been dismayed by the elaborate technique of Coffey will give the method a trial, commencing, if need be, with one ureter and two separate operations ; later, when they have learnt the technique, perhaps they will be persuaded to implant both ureters at one sitting ; for cases of carcinoma of the bladder and cervix or vesico-vaginal fistula are often in a pitiable condition of pain and suffering.

I feel that any technique which is simple and within the means of all surgeons, and especially those attached to a woman's hospital in the Tropics, will perhaps commend itself. The average time taken for double implantation has been one hour and a half.

It will give readers some idea of the frequency and, I might almost say, the malignancy of some of these fistulæ seen in the East when one relates that during the last ten years—that is, before we adopted transplantation of the ureters in desperate cases—my Registrar, Dr. R. R. Roy, has collected the case sheets of 104 consecutive patients admitted into this hospital with vesico-vaginal, uretero-vaginal, cervico-vesical, or urethro-vaginal fistulæ. He finds that out of these 104 operated upon, some of them multiple times, 55·8 per cent only were cured, 40·4 per cent failed, and 3·8 per cent died.

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RESECTION OF THE PRESACRAL NERVE IN UROLOGICAL CASES.*

By GORDON S. FOULDS, Toronto.

DURING recent years neurosurgery has been making rapid progress, and this is particularly true of surgery of the sympathetic nervous system. Little attention, however, has been paid to the application of this branch of surgery to the urinary bladder. Until quite recently our knowledge of the more exact anatomy of the nerve-supply of the bladder was sadly deficient. While most standard text-books of anatomy deal very inadequately with the subject, the French surgeons and anatomists have led the way, both in anatomical investigations and the application of anatomical knowledge to surgical intervention. The studies of Latarjet and Bonnet in 1913, and subsequent extensive work by Laux, together with clinical reports by Pieri and others, were largely disregarded until the work of Learmonth, who has done much to further clinical application of the anatomical data provided by the French investigators. In this more recent work many clinical observations have been possible, which lead us to a better understanding of the neuro-physiology of the bladder.

Micturition depends upon a very complex reflex, involving both the bladder itself and the internal and external sphincters. There is a diphasic neuro-muscular mechanism; the filling phase, which is involuntary, requires the relaxation of the bladder with contraction of the internal sphincter, while the emptying or detrusor phase is under voluntary control and involves relaxation of the sphincter and contraction of the bladder muscle. The bladder and its sphincters receive their nerve-supply from three sources—the thoracolumbar outflow of the sympathetic system, through the hypogastric nerves; the sacral autonomic system by the pelvic nerves or nervi erigentes; and from the somatic centres in the sacral part of the cord through the pudic nerves.
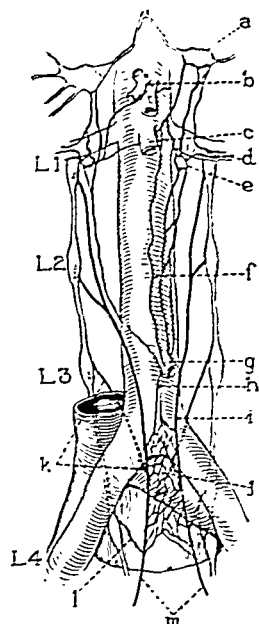
FIG. 59. — Connections of the presacral nerve. a, Semilunar ganglion; b, Cœliac plexus; c, Superior mesenteric artery; d, Renal plexus; e, Renal ganglion; f, Intermesenteric plexus; g, Inferior mesenteric artery; h, Middle root of presacral nerve; i, Left lateral root of presacral nerve; j, Presacral nerve; k, Branch from third lumbar ganglion; l, Branch from fourth lumbar ganglion; m, Hypogastric nerves. (After Laux.)

The hypogastric nerves have their origin in the presacral nerve described by Latarjet. This nerve is composed of three roots. Fibres from the first and second lumbar ganglia of the sympathetic chain on either side form the two lateral roots, which converge to unite in front of the body of the 5th lumbar vertebra ; they are pre-ganglionic and medullated. As these lateral roots converge they are joined by a third or middle root, which is made up of post-ganglionic, non-medullated fibres from the network of sympathetic nerves lying in close relation to the front and sides of the aorta known as the inter-mesenteric plexus (*Fig.* 59). These three roots unite in a varying manner to form the nerve bundle which descends parallel to the median line and crosses on the left common iliac vein, after which it follows the anterior surface of the sacrum. This bundle may be compact or separated, giving a fenestrated appearance. At a distance of 4 to 6 cm. the presacral nerve divides into the

FIG. 60.—Anatomical diagram of nerves to the bladder. (*After Learmonth.*)

two hypogastrics, which pass into the pelvis to join the hypogastric ganglia. The inferior mesenteric ganglion, so constant in lower animals and frequently referred to in writings on bladder physiology, is usually absent in man, and when present is represented by a thickening in the intermesenteric plexus about the root of the inferior mesenteric artery. The fibres from the lumbar ganglia descend, as previously described in the lateral roots of the presacral nerve.

The pelvic nerves are composed of fibres from the anterior primary divisions of the 2nd and 3rd or 3rd and 4th sacral nerves. These are pre-ganglionic medullated fibres and pass through the hypogastric ganglions to synapse with the nerve-cells of the ganglionated network of nerves in the bladder wall.

The third pathway is through the deep perineal branch of the pudic nerve which supplies motor fibres to the compressor urethræ muscle and sensory fibres to the deep urethra. This nerve has its origin in the 3rd and 4th sacral segments of the cord.

The filling phase of the bladder function is dependent on the motor impulses transmitted from sympathetic outflow by way of the presacral and hypogastric nerves. This nerve pathway transmits efferent motor stimuli to the internal sphincter and motor inhibitory impulses to the bladder muscle. It also carries afferent fibres to the bladder which transmit sensory impulses, notably those of overdistension of the organ with suprapubic discomfort. This was noted by Head and Riddoch, who demonstrated that pain set up by the ineffectual contraction of an .overdistended bladder in endeavouring to overcome an obstruction at its outlet is- referred to the 11th and 12th thoracic and 1st lumbar dermatomes, the somatic nerves of which. arise in

the same spinal segments as some of the pre-ganglionic connections of the hypogastric nerves. This nerve contains in addition vasoconstrictor fibres to the bladder. (*Fig.* 60.)

The emptying or detrusor phase of bladder action is under control of the pelvic nerves. which carry efferent motor stimuli to the detrusor muscles and motor inhibitory impulses to the internal sphincter. Afferent fibres from the bladder through these nerves represent a large proportion of the sensory fibres from the bladder.

Having reviewed briefly the nerve-supply to the bladder with reference to its function. I should like to report a case illustrating the application of some of these data.

HISTORY.—Mr. I. M., age 22. was admitted to St. Michael's Hospital on Dec. 2. 1930. complaining of loss of bladder control of fifteen months' duration. On Sept. 24. 1929. while intoxicated. he had fallen from a third floor window and sustained a compression fracture of the 2nd lumbar vertebra. Immediately following the accident he received hospital treatment and was placed in a plaster cast for three months. Neurologic examination at this time is said to have shown some paresis of the legs. but no loss of any localized movements. and sensory changes in a saddle-shaped area about the anus and scrotum in the area of the 3rd to 5th sacral segments. where there was an absence of sensation to pain and touch. There were no sensory changes in the legs or back.

He was unable to pass any urine and required to be catheterized regularly during the period in which he remained in the cast. Shortly after being allowed up he found that he was able to void by sitting down and pressing with the hands on the lower abdomen. He had no desire to urinate at any time, but felt pain suprapubically when the bladder became overfull. On such occasions the bladder could be felt as a pyriform mass in the lower abdomen, reaching nearly to the umbilicus. At first there was little or no difficulty in retaining urine, but later there was definite overflow incontinence at night or when careful attempts to empty the bladder partially during the day were neglected. These conditions continued up to the time of admission. Immediately after his accident there were no erections, but after several months the power of erection gradually increased until it had been fairly normal for over three months.

ON EXAMINATION.—The patient was a well-nourished white male. General examination was normal except for the presence of a mass in the lower abdomen extending to within an inch of the umbilicus and for a good deal of excoriation about the external genitalia due to dribbling of urine. After voiding as described above it was found that there was a residual urine of 28 oz. Catheterization was painful. The deep urethra was very sensitive.

Rectal examination showed poor tone of the rectal sphincters. The prostate was normal. Sensory changes were as previously noted. The bulbocavernosus reflex was present but not very active. Examination of the urine showed it to be acid ; specific gravity, 1022 ; albumin, trace ; sugar, negative ; microscopic examination showed the presence of a moderate number of pus cells. The white cell-count was 9200 ; blood-urea nitrogen was estimated as 12·2 mgrm. per 100 c.c. of blood. X-ray examination of the lumbar spine

showed a fracture-dislocation of the 2nd lumbar vertebra, while films of the urinary tract showed no evidence of stone or other abnormality. Cystoscopic examination was carried out under general anæsthesia. The internal sphincter was in spasm. The bladder contained 18 oz. of residual urine. Inspection of the bladder showed a general diffuse chronic cystitis. There was a moderate degree of trabeculation, seen particularly in the dome. Both ureteral orifices were normal in appearance and spurted clear urine. Ureteral catheterization with divided functional test and pyelograms showed no abnormality in the upper urinary tract.

DIAGNOSIS.—A diagnosis of 'cord bladder' due to injury to the lower segments of the cord (3–5S.) was made.

DISCUSSION OF CASE.—Taking into consideration the site of the injury, it is obvious that the sympathetic innervation of the bladder was not damaged. The hypersensitive posterior urethra, together with the weak but nevertheless present bulbocavernosus reflex, was taken to signify an intact pathway by the pudic nerves. The absence of any desire to urinate, and an inability to urinate satisfactorily, in view of the foregoing observations, corresponded with the localization of the injury and indicated serious interference with the sacral autonomic supply to the bladder by way of the pelvic nerves. However, in view of the observation that by taking time for relaxation of the sphincters and by the aid of the accessory muscles, partial emptying of the bladder was possible, and giving consideration to the fact that the power of erection had partially returned, it seemed reasonable to assume that there was not a total absence of impulses transmitted by the pelvic nerves, and that what really mattered was a lack of balance between the sympathetic and sacral autonomic innervation, the sacral autonomic normally being the positive or active force, while the sympathetic acts as a 'brake'. Now the 'brake' seemed to be too strong for the continued action of detrusor forces. Accordingly it was decided to interrupt the sympathetic innervation by resecting the presacral nerve and so remove its 'brake' action.

OPERATION (Jan. 5, 1931).—Under general anæsthesia a mid-line incision was made, extending from the umbilicus to the symphysis pubis. With the patient in deep Trendelenburg position the small intestines were packed up and the sigmoid to the left, thus bringing to view the bifurcation of the great vessels and the promontory of the sacrum. The posterior peritoneum was incised for about 1½ to 2 in. over this area and retracted gently. The two hypogastric nerves were isolated with little or no difficulty and severed just below the level of the promontory of the sacrum, and then followed up to their origin in the presacral nerve. This nerve was sectioned about ¾ in. above this junction, at a point where it crosses the left iliac vein. The posterior peritoneum was now closed, the intestines allowed to resume their former position, and the abdomen closed. An indwelling catheter was fastened in position and the patient returned to bed.

POST-OPERATIVE COURSE.—Immediately after operation the patient was given 0·12 grm. of acetylcholine twice daily intramuscularly. This drug is a specific stimulant of those structures innervated by the sacral autonomic system. In addition to this the bladder was washed regularly with 1–8000 potassium permanganate solution.

The indwelling catheter was removed on the tenth day. On the subsequent two days residual urine was found to be 15 and 12 oz. respectively. On the twelfth day a right-side acute epididymitis developed and no further estimation of residual urine was undertaken for ten days. At the end of this period residual urine was still 10 to 12 oz., so on Jan. 27 the indwelling catheter was again replaced for two weeks. After this period the residue was about 8 oz. Catheterization was done twice a day with gradually decreasing residue, until during March this was reduced to 1 or 2 oz. The man remained under observation until April 16, when he was dismissed from hospital with a residual urine of 2 oz.

When last examined, nine months after the operation, his chief difficulty was extreme constipation. When the constipation was very bad there was still difficulty with the bladder, at which time there was from 4 to 8 oz. of residual urine. Now, even if the bladder is allowed to become distended there is no suprapubic pain, proving the contention of Head and Riddoch that the sensation of overdistension is transmitted by the presacral nerve. It was also found that catheterization was not particularly painful, confirming the observation of Learmonth that the presacral nerve supplies some sensory fibres to the posterior urethra.

At the present time, under favourable conditions the patient can void fairly satisfactorily while standing, and on most occasions while sitting can empty the bladder to within 2 to 4 oz. He now has to depend entirely on the external sphincters for control. This is not always satisfactory at night, as urine frequently leaks during sleep. During the day control is generally satisfactory, but with over-filling of the bladder or fatigue, dribbling takes place. There is no question now of retention with overflow.

I think I may safely say, first, that this man is more comfortable; secondly, that residual urine has been reduced from 20 oz. or more, with distension and overflow—the usual condition—to 2 to perhaps 6 oz. Sphincter control is not well exercised, but I believe might be better with greater co-operation.

Pieri was first to point out the possibility of relieving the pain in some bladder lesions by section of the presacral nerve. In 1926 he performed the operation to relieve the distress from a painful cystitis. Viannay reported a further case the following year. Learmonth has made a preliminary report on four cases of this class. Three cases were suffering from a panmural cystitis, commonly spoken of as 'Hunner's ulcer', and the fourth was one of tuberculous cystitis, persisting nine years after a nephrectomy for tuberculosis, no tubercle bacilli being demonstrable in the urine at the time of the sympathectomy. Learmonth gives two reasons for attempting these operations: First, to relieve pain. This object was accompanied with a fair degree of success in three cases. Secondly, he is of the opinion that a more curative effect may be obtained by the vasodilatation brought about by the sympathectomy. The cases reported were too recent to make a definite statement as to what extent this hope was realized. Further, it cannot be claimed that all vesical pain can be controlled by presacral neurectomy.

Learmonth also reports two cases of sympathetic - parasympathetic

unbalance, in principle similar to my case.   One was due to a residual myelitis of the conus.   In both he obtained good results.

The number of cases in which this operation is applicable will naturally be small, but I believe it to be indicated in certain selected cases of unbalance between the sacral autonomic and sympathetic outflow, and in certain painful conditions of the bladder, particularly those associated with hypogastric pain with spasm of the internal sphincter.

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EIGHT LETTERS OF JOSEPH (LORD) LISTER TO WILLIAM SHARPEY.

By C. R. RUDOLF.

THESE eight letters written by Joseph Lister to William Sharpey were recently found by me in a second-hand bookshop in London.   The bookseller was unable to say where he had obtained them.   The first six letters submit Lister's ideas for Sharpey's criticism before offering his views to the Royal Society.   These views, being approved by Sharpey, were embodied in various physiological papers which became well known to the scientific world.   They led to Lister's recognition as an accurate and careful exponent of the processes of inflammation which had long exercised the minds of such thoughtful surgeons as Travers and Paget.

<div align="right">

11 RUTLAND GATE,
EDINBURGH,
16th November, 57.

</div>

MY DEAR DR. SHARPEY,

I have received my paper for revision prior to printing in the transactions, and along with Mr. Stokes[1] note was enclosed a report by Mr. Paget[2] on the paper.   Of this I enclose a copy in case you may not have seen it ; and now venture to trouble you for your advice on some points in Mr. Paget's suggestions, seeing that you are the " communicator " to whom he refers as the person through whom his suggestions should be conveyed to me : not to mention that your past kindness throughout this business, would have emboldened me to inflict myself upon you once more, even had you not occupied the position referred to.

First I must say that I am much impressed with Mr. Paget's care and candour in the matter, and, considering the position he occupies with regard to the subject, I think his remarks exceedingly handsome, and am much gratified by them.   Mr. Goodsir[3] also mentioned to me his having read the paper and endorsed it with his recommendation for publication, and he too spoke in terms, I fear, considerably flattering regarding the production.   I will now speak of each of Mr. Paget's suggestions and allude at the same time to Mr. Goodsir's remarks on the same points.

First as to omitting the account of the theories of previous writers. Mr. Goodsir also said, before he knew of Mr. Paget's report, that he thought the paper would be more classical without the historical sketch ; although at the same time he said he thought it " excellent ", supposing it necessary. But he did not think any such thing was required for a paper for the Royal Society.   I am quite willing on these grounds that this part should be

omitted, although, for the sake of the *general* reader, I should have preferred its being retained. I shall be guided entirely by you on the point. When writing this I do not know how much discretion is left to me. Mr. Stokes says " you will probably see reason to adopt " his (Mr. Paget's) " suggestions ".

As to the second part which Mr. Paget recommends me to blot out, viz., the discussion of vital affinities, etc., in the second section, Mr. Goodsir does not appear quite to agree with Mr. Paget. The former observed, " that is the opinion of a London man ". Now, unless you have seen the paper you could hardly form a judgment without my writing at greater length than would be expedient ; but I would ask you if there would be any objection to my retaining the mention of the observation of the absolute stillness of the blood in the capillaries and veins between the pulses in small frogs under the influence of chloroform. The fact that this state of things may continue for an unlimited time without any accumulation of corpuscles occurring in the vessels appears to me important, as the heart, though exceedingly weak, is then obviously the only cause of the movement of the blood. I would very much curtail my remarks on the bearings of the observation. It is not only in Edinburgh that persons think the vital affinities a cause of the blood's movement : witness Dr. Carpenter :[4] and here the opinion is rampant that [the] said affinities are the chief cause ? As to the discussion on the mode in which the constituents of the liquor sanguinis required by the various tissues for the purpose of nutrition find their way out of the capillaries, this I confess is very hypothetical and I am quite willing to expunge it. But it is not long, and, if there be no objection, it might be printed, and then if you thought well I would strike it out of the proof.

With regard to the expression about " paralysing " the concentrating and diffusing forces of the pigment cells, I feel seriously at a loss to know how to find a different mode of conveying my meaning. The facts are simply these. In the state of health the pigment cells exhibit the functions of concentration and diffusion of the granular portion of their contents under obedience to nervous influence. Also when a healthy limb is amputated concentration of the pigment (if previously diffused) takes place to the full degree and the pigment remains concentrated for some hours, after which a certain amount of diffusion again occurs, but if certain agents be applied to a portion of the web the pigment in that part ceases to manifest these functions. Not only is this the case when the limb is in connection with the body, but also when severed from vascular and nervous connection with the trunk. If the agent be applied to part of a dark limb just amputated the post mortem concentration does not occur in that part and again if the agent be applied when post mortem concentration is at the full, the subsequent diffusion does not take place in the spot operated on. In short, these functions of the pigment cells not only cease to vary in obedience to nervous influence, but also fail to exhibit the changes that ensue sooner or later after blood has ceased to circulate through the part : of which the post mortem concentration seems comparable to the post mortem rigidity of muscles. Now since I began to write this sentence I have been troubled with a difficulty that never occurred to me before, and which, I dare say, may be what Mr. Paget feels : and reflection upon this has detained this epistle two days. When speaking of

" paralysis of the concentrating and diffusing powers ", I convey the meaning that these powers not only cease to *vary* but cease to operate at all. Now I admit that this is by no means " proved " in my paper as it stands. There is good reason. I there state. to believe that the concentrating and diffusing forces are both in constant antagonistic operation. both being equally balanced except when either diffusion or concentration is occurring. Now all that I have shewn is that in the irritated part neither diffusion nor concentration occurs. and it may therefore be fairly argued that this does not prove more than that those forces cease to *vary* in *relative* amount under nervous influence, an effect which might result from paralysis of the extremities of the nerves, while the forces in the cells remained unaffected. But the consideration of all the facts with which we are acquainted regarding these pigment cells, including some which I have observed since the paper was written. seem to me to *prove* that the *cells* are affected independently of the nerves. and that this affection is one of paralysis. Now I am truly sorry to inflict this rigmarole upon you. but do not see how I can help it ; as I do not wish to run counter to Mr. Paget's opinion, and yet should be very sorry to give up the use of the term " paralysis " unless obliged to do so. You will remember that the German observers Brücke.[5] Wittich.[6] and Harless.[7] found that in the tree frog and the esculent frog concentration of the pigment resulted from the stimulus of light, from psychical excitement. from stimulation of the spinal cord, from stimulation of a branch of nerves leading to a particular portion of skin and from other stimuli. These observations tended to shew that concentration was the condition induced under the influence of stimuli applied through the nervous system. and that diffusion was the state with which the cells passed when left undisturbed by such stimuli. I have made similar observations on the frog of this country. which. however. is not nearly so sensitive to local stimulation. and I have also added the following fact lately. If I cut through all soft parts of the middle of the thigh except the artery, vein and nerve, the circulation goes on as usual and the leg retains the same colour as the rest of the body. But if I now cut through the nerve, the legs immediately become darker (if previously pale) and in a few hours the legs become black from full diffusion of the pigment, while the rest of the body may remain pale yellow. This very curious result is not *constant*, apparently in consequence of its being impossible to divide all the nervous filaments leading from the trunk to the limb, as proved by the fact that the arteries after a longer or shorter time regain their power of contraction, which is lost permanently if the brain and cord is removed. I have however induced this strange contrast of colour in three cases in which I performed the above operation. This experiment confirms the view that diffusion is the condition which occurs when nervous influence is removed.

An experiment by Wittich on the tree frog must be mentioned as illustrative of the same principle. He found that oil of turpentine when applied to a *moderately* dark tree frog caused the spot to which it was applied to grow pale, but after the oil had been removed the part not only returned to its former colour but became darker than other parts : the secondary diffusion of the pigment appearing comparable to the secondary dilatation of arteries after contraction from stimulation. So strongly did these facts impress the

German authorities that they considered them proof positive that concentration was the result of *contraction* of a cell and diffusion a relaxation of such contractile cell. Now I have shewn as you know, that this view is incorrect, as the cells do not change in form or size, but the pigment granules undergo changes of place in the fluid contents of the cells. And I have in the last few weeks made further observations on these curious processes, which shew that they are more complex than I had supposed. I have observed a cell in which slight diffusion was going on, and saw that the granules were moving in a very complex manner : one granule would be moving from the central dark mass of pigment while another was passing towards it. Sometimes a number of granules would start off together ; then stop and some of them return, etc., etc. Shewing that diffusion is the aggregate result of a complicated set of movements of granules some towards, others from the centre. Sometimes one saw a single granule start off from the central mass and return to it after a circuitous course, as shewn in the diagram [omitted], where (a) is the central black mass of concentrated pigment, and the dotted line represents the course of a granule. Such movements reminded me of what one sees sometimes within the cells of vegetables.

This description of the movements of the granules you will say is a digression from the argument, and so it is, but I wish to mention it to you, in order to let you know that I have made some further progress. But, however complex the action of concentration and diffusion be, there nevertheless remain the facts shewing that concentration is what occurs in the cells when stimulated through the medium of the nerves, and diffusion is that which takes place when the influence of the nerves is withdrawn. Hence it appears to me clear that, as an agent which induces inflammation causes the pigment cells to remain in the state in which it found them (say that of medium diffusion) however much they may vary in other parts, this effect is something more than paralysis of the extremities of the nerves could cause. Paralysis of the extremities of the nerves would [nay], *must* lead to diffusion unless the cells themselves were affected. Then I think we need have no doubt whatever that the *cells*, independently of the extremities of the nerves, are affected in such a way that they no longer act as usual in obedience to stimuli, in other words that the cells have the functions of concentration and diffusion *paralysed*.

What I should like myself, therefore, would be to retain the expression " paralysis of concentration and diffusion ", but to avoid the use of the expression paralysis of the concentrating and diffusing forces, or of the attractive and repulsive forces, as that implies what is incorrect regarding the physiology of concentration and diffusion, by giving the idea that during concentration the granules are all being attracted at once towards the centre, *and* vice versa.

One more point and one only : viz., regarding the publication of my supplement on the influence of the nerves on the arteries.

Mr. Goodsir has shown me a reference to Pflüger's[8] experiments on the large esculent frog, in which he found that division of the anterior roots of the spinal nerves causes relaxation of the vessels and stimulation of the same roots with galvanism causes the arteries to contract.

These are. I suppose. the experiments to which you had intended to refer me. but which I failed to find the account of. Had I been aware of them I dare say my experiments on this subject would never have been made : so that here. as in several other matters in this investigation. I have found myself anticipated. Yet at the same time I cannot but think some of my experiments deserving of publication. and it appears Mr. Paget thinks the same. but wishes me to make my results more precise. Now Mr. Goodsir agrees with me that experiments of the kind to which Mr. Paget alludes. viz., division of one half of the cord would be almost. if not altogether impracticable in the frog of this country. But I was aware of the possibility of making further observations regarding the seat of the nervous centre and have accordingly lately made some. In my paper I gave proof that a part of the cord as far forward as midway between the head and the caudal extremity does act as [a] nervous centre for the arteries after removal of the posterior half of the cord. What I felt might be further determined. but what I had not time to inquire into before the paper was read. was whether the posterior end in [the] tip of the cord is able to act as [a] nervous centre for the arteries after removal of the brain and the rest of the cord. Now this point I have since settled in the affirmative ; I have found that after removal of the brain and all the cord except the posterior *tenth* part (in length) the arteries after a while recover their contractile and varying power. Now this seems to me of importance. as it proves that the portion of the cord which Waller[9] and Budge[10] found to be the " ciliary " centre. etc.. etc.. the nervous centre for the dilatation of the pupil, and which they also described as the centre for the contraction of the arteries of the face. is not the *only* part of the cord which has this office. at least for the lower limb. Thus this experiment along with my former ones proves that the posterior half of the cord contains throughout its length parts which act as nervous centres for the arteries of the feet. And other experiments render it probable that the same function is possessed by a part somewhat further forward. Now I believe this is about as much as can be made out on this point from the frog. I have also several times verified lately an observation I had made before viz., that after removal of brain and cord the arteries remain *permanently* relaxed, while on the other hand the experiment I related in speaking of the pigment, viz., dividing all the soft parts visible in the thigh except the artery and vein, after which recovery of contractile power on the part of the arteries occurs if the cord is entire, shews very strikingly how very slender a nervous communication between the cord and the arteries suffices to enable the former to influence the latter.

Now I believe I am not likely ever to carry this investigation much further, nor do I believe much good could be gained by so doing, I mean in the frog of this country, and Mr. Goodsir thinks that it would be well for the results I have got to be published to avoid the chance of being anticipated. If, therefore, you approve. I would rewrite this supplement on the influence of the nerves on the arteries, introducing my additional matter, and somewhat curtailing the old experiments perhaps. But I do not think it would be necessary to alter it so much as to require it to be published as a separate paper at a later period. It might simply be stated

that some new matter had been introduced with this part, the new parts being specified.

I find that there is yet *one* point Mr. Paget suggests that the supplement in the anatomy and physiology of the pigmentary system should be published as a separate paper *before* the essay on inflammation. My chief objection to this is that the chief illustration, i.e., figure of the pigmentary affair illustrates also a part of the " essay on inflammation ". Also the last section of the paper is written as if the pigment subject was to be treated of in a supplement and I should prefer its remaining as such, if it might be so. This, however, is of small moment.

When must the paper be ready for printing ? I am intending to be in London at Christmas for a week, and if that time would not be too late, I should very much like to consult with you personally as to the best way of getting the illustrations drawn. I should think that for the pigment cells, which require much delicacy of execution, Tuffer West[11] would be as good a man as could be found.

My father seems to think that some of the sketches on inflammation almost require the same kind of simple *colouring* that W. Jones'[12] plates in the bats wing in the Philosophical Transactions have. Certainly the plates would tell their tale much better, and the colouring would be only a *very* simple matter, viz., red for congested capillaries, pale red for arteries and arterial capillaries, and pale purplish for veins.

I am really exceedingly ashamed of this letter. I only hope you will not quite give me up as a bore.

Agnes unites in kind regards to Miss. Colville[13] and yourself with

Yours very truly,

JOSEPH LISTER.

W. SHARPEY, Esqr., M.D., F.R.S., etc.

11, RUTLAND STREET,
EDINBURGH.
28. *Nov* : /57.

MY DEAR DR. SHARPEY,

You will be wondering what has come over me that I have not long since thanked you for and replied to your most kind and to me highly satisfactory letter. Nothing indeed could have been more welcome to me. The reason of my delay in answering it has been the pressure of my present engagements, which have hardly left me any available leisure, except at times when the mind was too much fagged for work of such importance. To proceed, then to business. I mean to act on your suggestions from first to last. The section " on the state of the tissues in inflammation " will, I believe, be very much improved by remodelling it on the plan you have suggested, viz : speaking first of the facts actually seen in the pigment cells, viz : the " arrestment " of concentration and diffusion and then *discussing the question of the* " paralysis ". That such " paralysis " really is present can, I think, hardly be doubted when the nature of the various agents which agree in *causing*

the " arrestment " is considered : for, while they differ among themselves exceedingly widely, they all have the common property of tending to destroy a part of the living body if maintained long enough in action upon it. It so happens, also, that the pigmentary tissue is a very delicate one, and is very apt to experience *permanent* injury without concomitant sloughing of the rest of the web. Thus if mustard be long enough applied, I find that the pigment cells begin to lose their sharpness of definition and appear to become disorganised. Similar effects on the pigment may also be seen in parts where mechanical violence has operated severely : and after some days the pigment in such parts becomes absorbed, and when once so removed is never replaced. There is one point on which you began to speak to me one evening at Millbank, but the conversation was in some way interrupted : viz. the change the blood experiences in the irritated part when its corpuscles become adhesive. You objected to my making use of such an expression as implied that the tissues in a state of health *exercise* an *active influence* over the blood in their vicinity, keeping the corpuscles from becoming adhesive. You said I might as well say that, because a man who was in good health when in a snug room grew rheumatic if exposed to the outside air or if holes were made in the walls, *therefore* the walls of the room *exercised* an *active influence* for good upon the man. I have often thought of that criticism and admit its applicability. But, at the same time, I must contend for activity *somewhere*, either in the blood or the tissues ; just as the *man* in the room is active though the *walls* are passive, so, if the tissues are merely negative in their operation in health, the blood must have a positively active power. The *facts* seem to be these. In blood withdrawn from the body, the corpuscles, both red and white, are more or less adhesive ; but within the vessels of a perfectly healthy part are quite free from adhesiveness. In the vessels of a part which has been acted on by some deleterious agent, (but short of actual disorganization or chemical decomposition) the corpuscles shew the same adhesiveness as when the blood is removed from the body. But this is not all. If the agent have acted so as to produce only a temporary effect on the tissues (as e.g. a powerful galvanic shock, which only temporarily " arrests " the changes of distribution of the pigment molecules) then as the tissues recover the blood also regains its natural healthy characters : the corpuscles previously viscid and closely packed, become non-adhesive again and are driven out of the vessels in which they had accumulated. Now I say this *recovery* on the part of the blood implies something active *somewhere*. We may either suppose that the *healthy* tissues exert a purely *negative* influence on the blood, which consequently does not suffer as it does when in contact with glass or any other dead matter, which must in such case be supposed to exert a disturbing influence leading to changes in molecular arrangement probably, an influence which would seem, very likely, synonymous with the " attraction of aggregation " which leads to crystalization, etc., etc.—or else we must suppose that glass, etc., has a negative influence and the healthy tissues an active one upon the blood. (I fear I am writing obscurely.) But supposing the former hypothesis be correct, and that blood in a healthy part has its corpuscles free from adhesiveness because *not* subjected to the action of disturbing forces such as are exerted on it by glass, sweet oil, atmospheric air, or paralysed tissues ; then, I say,

the fact that, as soon as the paralysed tissues recover, the corpuscles return to their original state of non-adhesiveness implies that the blood has an independent power of recovery from its injured condition, (compare the case of the rheumatic man) so soon as the tissues cease to exert the said disturbing influence common to dead matter, and resume their healthy negative condition. Now at first this seemed to me very unlikely : viz. that glass, sweet oil, the atmosphere, and paralysed tissues should be the *active* agents (for evil) and that the healthy tissues should be merely negative in their operation on the blood, but I have been inclined to look more favourably upon this view from considering the phenomena of *coagulation* of the blood. I think it quite needless to allude to the simple facts which, to my mind, prove clearly that no *chemical* theory yet given accounts for the coagulation of the blood : for I believe you are quite satisfied of the insufficiency of these theories. In some way or other the healthy tissues (which may remain healthy as *tissues* for some time after death) *do not* make the blood undergo the changes which lead to coagulation which ordinary dead matter (such as glass, air, etc., etc.) and inflamed tissues (paralysed more or less) *do* induce these changes in the blood. The principle I am stating is well illustrated by the speedy coagulation of the blood at rest in an aneurismal sac, in contact *not* with healthy tissues but with mere coagulated fibrine or inflamed tissue. The contrast presented by such a case with the length of time that blood will remain uncoagulated after extravasation among healthy tissues is very striking. Again the same thing is seen perhaps even more remarkably exemplified by the fibrinous masses that accumulate about calcareous atheromatous spicula projecting into the aorta or other large vessels, when the " dead " matter determines coagulation of fibrine notwithstanding the rapid movement of the blood (though to be sure only on the *lee* side of the projecting mass, when the blood is, I suppose, more or less quiescent). Now, such cases as these look to me very much as if the " dead " matter with its ordinary " attraction of aggregation " was the *active* agent, the healthy tissues having a merely *negative* influence on the blood. Also the bulk of the masses of blood which may remain long free from coagulation after death (e.g., that in the auricles of the heart) but which coagulate so soon as exposed to the influence of the external world, seems to me inconsistent with the idea of the tissues exerting an *active* influence on the mass of blood, and so preventing coagulation. Now, if the influence of the tissues is purely negative as regards prevention of coagulation, this is a strong argument in favour of their being also negative as regards prevention of adhesiveness of corpuscles. Hence I mean to avoid the expression to which you objected.

I am glad to learn that you have verified the observation of the intermitting flow in the tadpole's gills. I alluded to Spallanzani's[14] observation in a note to my paper [p. 221], but did not lay very great stress upon it until I should be able to learn whether there is any *recoil* observed between the pulses. Can you inform me on this point ? In the case of the young frog under chloroform there is certainly no recoil, only absolute quiescence, between the pulses. The case of the tadpole's gill seemed to me to differ from the adult frog's web in this respect that, as I suppose, there is only one auricle in the young tadpole as in the fish, or at least the right auricle is not brought into action for the

gills, and therefore the ventricle has to overcome the resistance of two sets of capillaries, the branchial and systemic, when driving the blood into the aorta. Now, it seemed to me that the resiliency of the elastic aorta beyond the gills might give rise to a recoil in the branchial capillaries, in which case the observation, though it would shew abundantly the superiority of the force of the heart to any other forces that might be conceived in operation, would not *prove* that no such forces did exist as causes of movement. With regard to the adult frog with weakened heart I have some doubt whether sufficient care has been taken to discriminate between the interruptions of flow due purely to weakness of that organ and such interruptions due partly to an unhealthy state of the blood with somewhat adhesive corpuscles. For I know that, unless attention has been specially directed to the matter, a very decidedly abnormal state of the circulation as regards the excess of slowly moving corpuscles may pass unnoticed.

One point I would ask your advice about is how the section " on the influence of the nerves upon the arteries " had better appear : whether as a supplement, as it now is, or as a separate paper. Mr. Paget says it should appear " if at all, as a separate paper ", and Mr. Goodsir seems to think it forms such a distinct subject that it might be well to have it separate. In this matter I will be guided entirely by you. This only I would say, that if it be a separate paper it should appear, like that in the " pigment business " and in the same volume of the transactions, and, if possible, immediately before the others, or intermediate between them. I feel, for my own part, very indifferent about this, though I quite agree now that the *pigmentary* supplement will be better as a separate paper.

And now to reply to your question as to what things cause *concentration* and what *diffusion* of the pigment. First I would observe that there is this marked difference between the occurrence of concentration and diffusion under the influence of local applications, viz., that concentration occurs not only in the spot acted on, but in a greater or less area round about : in this respect concentration agrees with the contraction of arteries which occurs *round about* a part irritated, and both are obviously *reflex* phenomena, effected through the medium of the nervous system. Diffusion on the other hand occurs, as induced by a local application, only in the precise spot on which the irritant acts, e.g., beneath a little piece of mustard put on part of the web, but not at all beyond the limits of the mustard. Hence this diffusion is clearly due in some way to direct action of the irritant upon the part of the web on which it lies, and is not a reflex phenomenon. To this statement you will say I mentioned a contradictory fact in my last letter, viz., the *secondary* diffusion that Wittich observed after applying oil of turpentine to a moderately dark frog. This *secondary* diffusion *is* doubtless a reflex phenomenon, and is comparable to the secondary relaxation of arteries that occurs round about an irritated part. But I have never witnessed this *secondary* diffusion and possibly never shall in *our* frog, whose skin is far less sensitive to direct irritation than is that of the tree frog on which Wittich operated. Thus, oil of turpentine produces no primary concentration when applied to our frog, and its only effect on the pigment is to produce " arrestment " of concentration and diffusion. So little sensitive, indeed, is our frog to direct *stimulation,*

variance with what I should have expected was that of the ciliary action going on in water impregnated with carbonic acid, for I had found that "aerated water" which I am assured contains nothing but carbonic acid, causes arrestment of the pigmentary changes. But then it occurred to me that very probably the carbonic acid might arrest the ciliary action also if *long enough* applied, just as the pigmentary arrestment and development of inflammatory stagnation of the blood require a considerable time of the action of the carbonic acid. And so it turns out. I put four bits of mucous membrane from different parts of the mouth and œsophagus of a frog in a glass of cold water, and four corresponding bits in a bottle of aerated water, and left both for about two hours. At the end of that time I found ciliary action in every specimen from the plain water, and not a trace of such action in any of those from the aerated water, although the cilia were distinct enough. There was, however, a great disposition to desquamation of the epithelium cells in the latter (from the carbonic acid water). I have since repeated this experiment on another frog with the same result. So far then, as I have tried, any agent that causes pigmentary arrestment when applied to the web causes also ciliary arrestment, and that not destruction of tissue, but suspension of function. Now there can, I conceive, be no doubt that the cilia, or the cells which bear them, are truly *paralysed* in these experiments, i.e., unable for the time being to act, and it is also *perfectly* clear that the effect is independent of nervous or any other influence from the rest of the body of the creature, for, as everyone knows, and as I saw abundantly in these experiments, the cilia continued to act after cells have been separated from the mucous membrane, *unless* you act upon them with one of these deleterious agents. The effect then is clearly a *direct* effect of the irritant on this form of extra vascular tissue. Do you not think that these simple experiments very much serve as experimenta crucis as regards the *nature* of the arrestment which irritants produce on the tissues, viz., that they paralyse. You see they do not in any way clash with yours, but it so happens that my former observations led me to try a set of agents which you had not employed. I see you found aether arrest the ciliary action; and I do not doubt it would excite inflammation if applied to the web. Hoping you will excuse me for thus trespassing again on your attention, I remain, with kind regards to Miss Colville, in which Agnes[15] unites,

<div style="text-align: right">Yours very truly,
JOSEPH LISTER.</div>

W. SHARPEY, Esqr., M.D., F.R.S., etc., etc.

<div style="text-align: right">11, RUTLAND STREET,
EDINBURGH,
28th June /58.</div>

MY DEAR DR. SHARPEY,
    I heard the other day from Mr. Stokes to whom you forwarded my last to you, and his reply was in all respects quite satisfactory. Many thanks to you for procuring me the information it contained.

And now I must needs trouble you with another question : viz., whether a paper written now could have its abstract appear in the next number of the " Proceedings ". I remember noticing in the number which contained the abstract of my inflammation paper that abstracts were given of some papers received after the Royal Society had ceased to meet for the season, some indeed as late as August.

I have been lately making some experiments in the so-called " hemmungs nervous system ", a subject in which I have felt much interest from its connection with the origin of inflammation through the medium of the nervous system, as when the lungs inflame through long continued coldness of the surface of the chest. Some of my experiments with tepid water on the frog's foot had shewn that in the case of the *arteries*, relaxation of the muscular fibres in consequence of the application of an irritant to the skin is the result of the nervous centre (the spinal cord or those parts of it which preside over the arterial contractions) being thrown into a state of inaction. The very same stimulus whose *mild* action led to *action* of the nervous centre (with consequent contraction of the vessels) produced inaction of the same nervous centre when applied in a more intense degree. This was *proved* by the fact that the dilatation which occurs after contraction as the results of the application of tepid water of pretty low temperature gives place to contraction on re-application of water of the same temperature. Now—if the secondary dilatation was the result of any *action* of the nervous apparatus in consequence of the stimulus that dilatation would have been increased, instead of giving place to contraction, in repetition of the same stimulus. It is therefore clear that this secondary dilatation is the result of inaction of the nervous apparatus concerned. But it is quite impossible to draw the line between the dilatation which succeeds to contraction in consequence of a mild irritant, and that dilatation which occurs immediately, or, if you choose to assume it, after a " momentary contraction," after a stronger irritant, or the same irritant more strongly applied. Hence these experiments with tepid water lead to the remarkable conclusion that—given an afferent nerve, a nervous centre, and a motor nerve presiding over the contraction of unstriped muscular fibres— a very mild stimulus applied to the afferent nerve may (e.g., in the case of the arteries) lead to *action* of the nervous centre followed by muscular contraction, but a somewhat stronger stimulus applied to the afferent nerve may cause *inaction* of the nervous centre, and consequent muscular relaxation.

Having got this analogy to proceed upon I could hardly doubt that the same thing occurred in the so-called " hemmung system ", viz., that if a mild enough stimulus were applied to the nerves, the contractions of the intestines and action of the heart would be increased instead of arrested. In order to put my ideas to the proof of experiment, I employed a coil battery, acting so feebly as to be only just appreciable to the tongue placed between its poles, and I have found the result such as I anticipated.

1st, as regard the intestines. I found the best mode of proceeding was to remove the skin and two layers of muscles from the abdomen of a rabbit, leaving the peritoneum and one layer of muscles, which are quite transparent enough to enable you to see any movements of the intestines, without the complication that the action of the air upon them involves. I found that,

when a coil battery with its poles attached to the 6th and 12th dorsal spinous processes was put in action in the excessively mild manner I have mentioned, the writhing movement of the intestines became invariably greatly aggravated as long as the galvanic currents were acting on the cord, but as soon as the circuit was broken the former condition returned. But I also found that if the rods of soft wire were put into the helix, the " hemmung " action came into play and the intestines became relaxed and motionless : yet the action of the battery was very feeble, not even *unpleasant* to the tongue, when the rods were in. I also found that mechanical irritation of the spinal cord with a fine needle in the " dorsal region " (the same kind of irritation which I had always seen followed by contraction of the frog's arteries) produced increased contractions of the intestines. This I find from a paper to which Mr. Goodsir has referred me in Henle and Pfeufer's Zeitschrift for last year, has been observed also by a German, Spiegelberg[16] of Göttingen ; and his observations are particularly satisfactory as they were only incidental to the subject of his enquiry, viz., the movements of the uterus, and made before he heard of Pflüger's " hemmung " experiment.

I have also found, and this is so far as I know a new observation, that the increased peristaltic action which comes on after death in consequence of the cessation of circulation, stops when the " hemmung " action of the battery is set on. If you wait many minutes after death you will not see this, as the spinal cord soon loses its vital powers. But an experiment of the same kind may be repeated almost as often as you please in the following way. Tie with a fine needle and thread two or three principal arteries in the mesentery leading to about four inches of the intestine of a rabbit (the intestine lying upon a plate covered with fresh olive oil) and very speedily the movements, analogous to the post mortem movements, occur in this part and nowhere else. Then set on the strong action of the battery, whose poles have been previously attached to the spinous processes, and immediately the movement ceases. This experiment may be several times repeated. Of course it is necessary to tie the little arteries very delicately so as to avoid as much as possible the little nervous filaments. This experiment is of double significance ; first it proves very clearly that the increased peristaltic action that ensues on death is due simply to arrest of circulation in the part, for the " hemmung," on action of the battery, proves that the nerves have not been injured and therefore that the increased peristaltic action was not the result of injury of the nerves. 2nd, it proves that the arrest of the circulation does not *necessarily* produce peristaltic action, but that this post mortem peristaltic action, like that which occurs during life, requires that the nervous apparatus *in* the intestines should be in working order. I say the nervous apparatus *in* the intestine, for it is of course well known that the post mortem peristaltic action goes on after the mesentery has been cut off close to the gut. The existence of such a nervous apparatus distinct from the contractility of the muscular fibres is quite evident. from the observation of Pflüger, which I have confirmed, that during the " hemmung " which results from powerful galvanic action through the cord, the intestines contract *at* any part irritated, though the movement does not extend to other parts ;. the muscular contractility is unaffected although the co-ordinating apparatus *is* thrown

out of action. I have added further proof of the distinctness of these two functions, muscular contractility and co-ordination of movement, in the observation that the faculty of contracting *locally* on irritation remains in the intestines of a dead animal for some time after the power of peristaltic action has ceased : the nervous apparatus dying before the muscular. Mr. Goodsir has, since these observations were made, pointed out to me Meissner's[17] discovery of ganglia in the submucous tissue of the intestines, recorded in the last part of the former series of Henle and Pfeufer's Zeitschrift (1857); shewing the mechanism by which, no doubt, this co-ordination is effected.

Again I have shewn that the ordinary peristaltic action which occurs during the life of the animal is affected by the apparatus within the intestine : for if you cut in the mesentery the fine nervous branches leading to a certain portion, say three inches, of the intestine, taking care not to injure the blood-vessels you find that the peristaltic action goes on as in adjoining parts quite unaffected by the operation, whereas, if the peristaltic action was produced through the influence of the solar plexus or other nervous centres distant from the intestine, it should cease when the nerves in the mesentery are divided. Then, to prove that the nerves leading to the piece of intestine have been thoroughly divided, you have only to set on the strong galvanic action through the cord, when you see perfect rest and relaxation of all parts except that to which the nerves have been cut, and that continues to move just as before. This is a very striking experiment, and further proves that the nerves leading from the cord to the intestine must be in a state of *action* as far as the intestine in order to produce the " hemmung ". From these and other observations with which I need not bore you at present, it follows quite clearly that the " hemmung " of the movement of the intestines is produced by the action of the nerves leading to the intestines upon the nervous centres contained in the gut, but that this is not the only effect of the action of these nerves, a milder action of the same nerves increasing instead of preventing the action of the co-ordinating nervous apparatus of the intestine.

Next as regards the heart, I find that if after dissecting the vagus of one side from surrounding connections without injuring it by pinching or otherwise, and isolating it by a bit of glass placed between it and surrounding tissues, you apply to it the poles of the coil battery tipped with fine silver wire, the battery acting so feebly as scarcely to be appreciable to the tip of your tongue, the beats of the heart are increased in frequency instead of diminished, but that if the rods of the soft wire are put into the helix the action of the heart becomes diminished in frequency, gets irregular and at length stops. This shews, as in the case of the intestines, how very gentle a stimulus must be used in order to produce *increase* of action of the ganglia presiding over the action of the viscus. This, however, is quite consistent with the great effect produced by slight stimuli in many other cases : e.g., when a touch of the skin at one point induces horripilation over a large extent of surface. But had I not been led to do so by the analogy of the arteries, before alluded to, I should probably never have dreamed of using a battery so excessively feeble in action. I see that here too I have been anticipated ; for Spiegelberg alludes to experiments by Schiff[18] recorded in Vierordt's archives (which I have not been able to get) by which he is said to have shewn that a

gentle stimulus applied to the vagus promotes, instead of arresting the action of the heart.

One experiment mentioned by Pflüger in his book on the "hemmung nerven system" (1857) seemed strongly in favour of a constantly operating controlling action on the part of the vagus over the heart's contractions; viz., That division of the vagi increased the frequency of the heart's beats: even doubled their frequency. At the same time it is said that this has not been seen in *frogs*, only in mammalia. On this subject I made the following experiment. The abdomen of a rabbit having been for ¾ of an hour open and the intestines out upon a plate covered with sweet oil, the beats of the heart not having been as yet at all affected as far as I could judge, but being about 42 in 10 seconds, I exposed both vagi in the neck, taking care not to pinch them, but isolating them so thoroughly from the sympathetic that I could at any time divide them with facility without injury to surrounding parts. Having thus counted the pulsations in a coiling artery of the mesentery and found them 42, I rapidly divided both vagi in the neck, each with a momentary act with a pair of fine scissors, and about a minute and a half after the operation, found the beats 40 in ten seconds. A minute or two later the beats were 38, and so on, the pulsations gradually diminishing till ¾ of an hour after the operation they were 28. When they had fallen to 32 I pinched one vagus with forceps, and within about a minute of the commencement of the pinching, and while the irritation was still continued, the pulsations were again 40 in the 10 seconds. Similar results followed from the irritation on subsequent trials, but in a less marked degree. Finally after the chest had been opened and respiration had ceased, I found that the same irritation which had before increased the heart's action, viz., pinching the vagus, diminished the number of beats: the ganglia of the heart being, I suppose, so feeble as to be thrown into diminished action by the same stimulus which had previously induced them to act more.

In this experiment the fallacies which may arise from opening the chest were avoided, and it proves of itself quite conclusively that there is not *always* in operation a controlling action of the vagus over the heart's action. On the other hand I have observed in the case of the intestines that *invariably* during a prolonged struggle of the animal the peristaltic actions cease; which seems to show that natural and normal actions of the nervous system *may* induce the "hemmung".

If you can find time to set my mind at rest as to whether I shall prepare this paper for the Royal Society or do it in a shabby way for the *Lancet* you would do me a very great kindness. Of course in a case of this kind, where so many persons are working at the subject, it would be very desirable to have a notice of this paper early: and the proceedings of the Royal Society would of course be the best medium.

I need scarcely say that if the paper does go to the Royal Society, what would please me best of all would be for it to be thought worthy of a place in the Transactions as well as the Proceedings.

Agnes joins in kind regards to Miss Colville and yourself with

Yours very truly,

JOSEPH LISTER.

WILLIAM SHARPEY, Esq., M.D., Sec. R.S., etc.

11, Rutland Street,
Edinburgh,
7th July /58.

My dear Dr. Sharpey,

I fear my last letter to you must have arrived at a time when you were very much occupied, or else that you were very sceptical as to the trustworthiness of my observations. I suspect that the latter was the truth ; for I admit that, considering the difficulty of the subject, and the variety of statement by different observers hitherto, anyone who goes to work at the " hemmungs nervous system " with a preconceived idea to support may be justly suspected of being unconsciously influenced, like the table turners, by his prejudice, and made to see things as he wishes rather than as they are. But I have endeavoured to bear fully in mind this tendency of the human mind, both in this and many previous observations, and, I trust guarded against it. Since I wrote to you I have made some additional experiments, two of which I will mention, for the sake of making you give some credence to those which I before related.

(1) It has been stated that division of the vagi produces in mammalia increased action of the heart. This, if true, would be strongly in favour of a constantly operating controlling action on the part of the vagus. I found it stated in Pflüger's book, however, that this effect had not been observed in frogs : and this seemed to me to throw doubt upon the alleged fact in mammalia. Accordingly I tested it by experiment. In 4 rabbits and a calf I have divided both vagi ; I need not mention the details of the precautions used to avoid fallacy but may simply state, that in no single case was increase of the number of beats the result of division either of one or both nerves. Hence, even on the unlikely supposition that these were exceptional cases, these facts prove that there is not any *constant* controlling, i.e., checking influence exercised by the vagus over the heart's action.

(2) I had observed accidentally in one case in which I had been experimenting on the intestines after Pflüger's method (the poles of the battery being fixed to the 12th and 6th dorsal vertebræ) the vagi also having been both divided, the heart's action having grown very feeble was greatly improved by the application of galvanism to the cord. This shewed that the spinal system could influence the heart's action *otherwise* than through the vagi ; and the same thing was proved by the frequently observed increase of the heart's action when the animal struggled, in cases where both vagi had been divided. Now it occurred to me that, if my view were correct, viz., that the nervous centres in the heart, intestines, uterus and probably other internal organs, were liable to " hemmung " from a little increase of action of the nerves (leading from the cord) whose *mild* action made these nervous centres more active ; then the same kind of effects ought to follow from the irritation of other nerves proceeding from the cord to the heart as from irritation of the vagus ; viz., increase of action with excessively mild stimuli, and " hemmung " with stronger stimuli. To test this I fixed the silver wire poles of my battery, one to the 6th dorsal spinous process, and the other to about the 2nd or 3rd *cervical* spinous process, so as to transmit the currents through

that part of the cord which sends off branches, through the sympathetic, to the cardiac plexus. I then found that when the action of the battery was the feeblest that I could recognize with the tip of my tongue placed between the fine silver wire poles, the heart's beats were increased by the galvanism so long as it continued in operation the number of beats in 10 seconds being raised 2 or 3, time after time, but after the circuit was broken and the action of the galvanism discontinued, the heart's beats fell to 5 or 6 beats below what they were before the galvanism was employed, and then after about 2 minutes regained their former number, e.g., in one case the beats were 37 or 38 before the galvanism was applied, 40 to 42 during the minute or two that the galvanism acted, then after it had ceased fell gradually but rapidly to 33, and then rose again to 37. These effects were observed time after time without exception; the increase in the beats during the galvanism being most marked at the commencement of the experiments, but the fall immediately after the cessation of the currents being as constant as it was striking. I could not but wonder that a degree of action of the battery so very faint should produce such effects. Then, when the rods of soft wire were put into the helix, or a little more acid into the jar, the action of the battery being still quite mild as felt by the tongue, the effect of connecting the circuit was to produce " hemmung ", i.e., depression of the number of beats by about half a dozen *during* the action of the galvanism, and this time after time. I have experimented on two rabbits in this way, and in both found the results clear and satisfactory for a long time (about 2 hours), after which the galvanism failed to produce either increase or diminution of action of the heart ; whether it was applied feebly as before or so powerfully as to elicit sparks from the parts of the tissues touched by the poles. It was quite evident that at these times the nerves concerned in conducting the stimuli to the cardiac ganglia were exhausted : and anyone repeating such experiments must be very careful not to use a strong action of the battery at first, or the exhaustion may occur very speedily, and then no effects upon the heart will shew themselves. I should mention that the counting of the beats of the heart with precision was greatly facilitated in my last experiment (done yesterday) by removing a portion of skin and pectoral muscle from the præcordial region of a half-grown rabbit, when the heart's action could be seen with the utmost ease through the transparent intercostal muscles and pericardium.

So this experiment confirms the general doctrine of the liability to diminished action of the part of the nervous centres seated in the viscera, in consequence of increase of the action of the nerves leading from the cord to them : and further shews how extremely delicate must be the stimulus applied to the cord or the nerves in order that the *increased* action of the nervous centres aforesaid may be developed. Also it shews how liable the nerves leading from the cord to these nervous centres are to lose their function in consequence of long continuance or excess of the stimulation. I expect to be in London at the end of this month, and, if you thought fit, would bring with me an abstract of a paper on this subject : for I really think I have now got facts enough to warrant me in publishing. If you do not approve of this plan, I would send a paper on the subject to the *Edinburgh Medical Journal*

for next month. The paper would have to be differently got up according to the medium in which it was to appear, so there seems no time to lose. May I then ask the favour of an early reply.

Yours very truly,

JOSEPH LISTER.

WM. SHARPEY, Esq., M.D., Sec. R.S.

11, RUTLAND STREET,

EDINBURGH,

28th Nov. /58.

MY DEAR DR. SHARPEY,

I am much obliged to you for the information conveyed in your kind note received this morning. I dare say my third (and last) paper might have been at the printer's before now, had not a recent letter from Tuffer West made me give up hope of getting it into the next part of the Transactions. Now, however, you may be sure, I will not waste a minute, for I am very desirous to get the job off my hands. Half the last paper is already fit for the press, and I will send it at once, and get the rest done in a few days.

One advantage this delay has had ; viz., it has shewn that I was wrong, and you were right after all about the matter of the coagulation of the blood. Your view was that the reason of coagulation on removal from the vessels or on injury or disease of the vessels was a difference of *surface* in the surrounding objects. Mine had been that surrounding objects were neutral as regards the blood, but that the tissues of the vessels were active in preventing any tendency to escape of ammonia, etc., etc. My reason for this view was that I found that air freely admitted into the vessels along with the blood had no effect in causing coagulation. Hence, as exposure to air has been supposed by so many physiologists to have great effect in causing coagulation, I rashly inferred that because the atmosphere was neutral in its effect on blood within the vessels, therefore *all* dead matter was so, *solid* as well as gaseous. But by some recent simple experiments upon sheep's feet I have found that if a piece of fine silver wire, wax, hair, glass, etc., etc., be introduced into a vein, the fibrine deposits upon the solid and, if the mass be considerable, the clot may be large ; although mere surrounding of a vein without introducing any extraneous solid does not cause coagulation except in the mouth of the wounded vessel. From these and other facts, which I must not detain you now with detailing, it seems to follow that the lining membrane of the vessels (and probably other tissues also) differs from any ordinary solid substances I have yet tried in being *neutral* as regards the tendency to cause deposition of the fibrine from solution, while silver, wax, etc., and the tissues themselves when diseased, attract the fibrine or in some way determine its precipitation, probably as a crystal does a solid in solution. Outside the body it appears that a certain small amount of ammonia is necessary to keep the blood fluid, but within the vessels the ammonia seems not at all essential to the maintenance of the fibrine in solution. For it is easy to prove that most of the ammonia, if not all of it, escapes from the

blood in the vessels of an amputated sheep's foot, when the foot has been kept a few hours or when a vein has been wounded. Under these circumstances the blood let out from a vessel coagulates immediately, so that I have seen evidence of the commencement of the process within 5 seconds of the shedding of the blood. This rapid coagulation must I imagine be attributed to escape of ammonia ; yet if the blood thus devoid of ammonia or nearly so be left in the wounded vessels it will remain fluid for any length of time, hours or days.

I have been induced to write you a much longer account of this matter than I intended, but I hope you will excuse this. Mr. Syme has returned safe and sound, and apparently well pleased with the results of the council meeting. He gives a good account of your health.

Agnes unites in kind regards to Miss Colville and yourself with

Yours very sincerely,

JOSEPH LISTER.

Excuse great haste.

WILLM. SHARPEY, Esqr., M.D., Sec. R.S., etc.

(To be continued.)

SHORT NOTES OF
RARE OR OBSCURE CASES

A TUMOUR OF THE MOLAR GLANDS.

By C. GRANTHAM-HILL,

SENIOR SURGEON, KHARTOUM HOSPITAL.

THE patient, a Sudanese woman of about 50 years of age, complained of a large and painful swelling on the left side of the face (*Figs.* 61, 62). She noticed it first as a small swelling near the left angle of the mouth a year previously, since when it had steadily increased in size, and had, during the last two months, become painful. On palpation the tumour was lobulated and elastic and was not attached to the jaws or parotid gland, being freely movable. It was thought to be a sebaceous adenoma.

OPERATION.—After preliminary removal of some carious teeth and cleaning of the mouth, the inferior dental nerve was blocked with novocain inside the mouth, and the growth surrounded by a subcutaneous infiltration block of the same solution. The growth was easily dissected out, following a well-defined capsule, which was not adherent to the surrounding tissues, excepting to a small area of buccal mucous membrane near the opening of Stenson's duct. This piece of mucous membrane was removed along with the tumour without interfering with the duct. Enough skin was saved to cover the wound. Post-operative recovery was uneventful except for a small fistula which formed at the point at which the mucous membrane had been removed, and which closed spontaneously. (*Fig.* 63.)

PATHOLOGY.—I am indebted to Dr. T. F. R. Hewer of the Welcome Tropical Research Laboratory for the following report :—

" The tumour weighs 530 grm. and measures roughly 12 by 11 by 8 cm. It is a coarsely lobulated mass whose borders are well defined and encapsulated : the overlying skin is thinned over some of the larger bosses of the tumour, but even here there is no invasion beyond the capsule, and the skin can readily be stripped off. The tumour is firm and resilient, and on section it offers considerable resistance to the knife. The cut surface, after fixation, shows irregular trabeculæ of dense connective tissue, partly hyaline and partly opaque, passing from the capsule to the centre of the tumour mass. The parenchyma consists of small irregular lobules of pinkish grey or white colour, lying within a fine meshwork of connective tissue. At the centre of the tumour is an area of hæmorrhage in which the parenchyma has been replaced by masses of soft friable colloid and blood-clot ; nearer the periphery there are also a few chalky or caseous patches of necrosis. There does not appear to be any deposit of calcium, nor is there any sign of cyst formation.

" Microscopical examination of a section reveals a solid structure of large polyhedral epithelial cells arranged in lobules circumscribed by fine septa of connective tissue; these cells have a granular cytoplasm and a large dark-
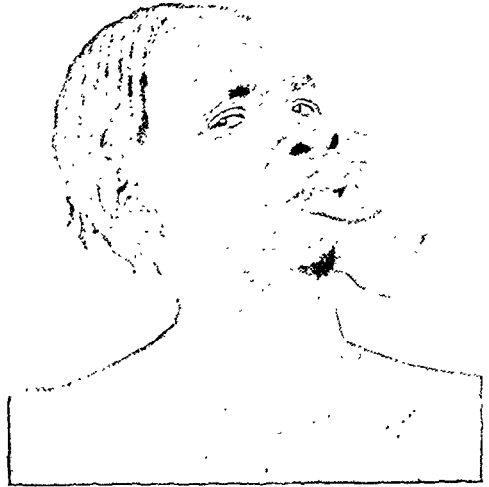
Figs. 61, 62.—Tumour of the molar glands.

Fig. 63.—Appearance of patient after operation.

staining nucleus. There are occasional areas of necrosis in the centres of the lobules, and, in other parts, clefts have arisen which give an appearance of acini, but in reality there is no suggestion of such a formation. All parts of

the section contain the same type of cell ; there is no sign of duct formation or of any other tissue. The tumour would seem to be a solid adenoma of a simple salivary gland." (*Fig.* 64.)

Comment.—There is reason to believe the tumour to be one arising from the molar glands, described in *Gray's Anatomy*[1] as being, " four or five, placed between the masseter and the buccinator muscles around the distal end of the parotid duct ". C. E. Shattock[2] (1929) says that these may give rise to rare tumours similar to mixed parotid tumours. but in front of. and quite separate
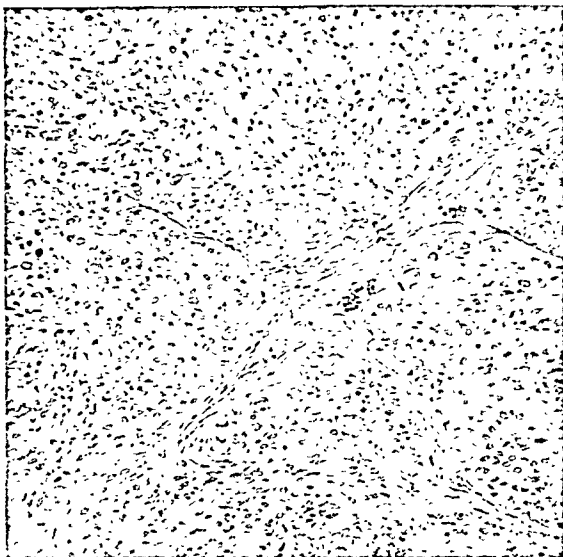
Fig. 64.—Microscopical appearance of tumour. (× 100.)

from, the socia parotidis ; that the tumour may spread through the capsule and infiltrate the surrounding tissues, though at first movable or rounded or lobulated ; and that it may extend forwards almost to the angle of the mouth. Lenormant, Duval, and Cottard[3] (1908) collected ten cases of tumours of this type, some occurring on the lips. Some contained bone and cartilage, some were cystic, and some solid. None were malignant, and nearly all occurred in young people.

I am indebted to Dr. H. M. Elliott for sending me the case ; to the Director, Sudan Medical Service, for allowing me to publish it ; and, particularly, to Professor Arnold K. Henry, of Cairo, for references in the literature.

REFERENCES.

[1] *Gray's Anatomy*, 1918 ed., 1046.
[2] SHATTOCK, C. E., *Handbook of Surgical Diagnosis*, 1929, 461.
[3] LENORMANT, DUVAL, and COTTARD, " Les Tumeurs mixtes de la Joue et des Lèvres ", *Rev. de Chir.*, 1908, xviii, 1.

THREE ENTEROLITHS IN A SINGLE COIL OF JEJUNUM.

By R. E. KELLY, C.B.,

SENIOR SURGEON, LIVERPOOL ROYAL INFIRMARY.

STERCOLITHS, although common in domestic animals, are somewhat rare in man. They are generally single, may attain the size of a golf ball, and usually cause intestinal obstruction in the small bowel. A rounded impediment of this kind may easily be mistaken for a gall-stone, from which, indeed, it can only be distinguished by chemical analysis.

Their cause is obscure. The present case would seem to show that they may be precipitated in a dilated and hypertrophied coil of intestine proximal to a stricture. Further, the case is interesting as showing three enteroliths all lying in a single coil of jejunum, and all imprisoned between two strictures in the gut so that they could pass neither onwards nor backwards. The result was that by their ball-valve action on the distal stricture this particular coil of jejunum had become so dilated and hypertrophied that it resembled a second stomach. The coil was about 10 in. long by 2 in. wide.

The three enteroliths were of varying sizes, so that it is probable that they were not all formed at the same time. Perhaps the damming back of the intestinal contents by one enterolith favoured the deposition of the other two. In this patient their formation probably took many years.

HISTORY.—Twenty years ago I operated on a girl of 12 for an 'acute abdomen'. The diagnosis had been appendicitis. This was a mistake, for the appendix was normal. There was a suppurative pelvic peritonitis due to a perforation of a tuberculous ulcer in the jejunum. The perforation was stitched up and the pelvis drained. She made an uninterrupted recovery and for ten years was quite well.

For the past ten years she has had attacks of vague abdominal pain. The pain bore no relation to food, had a sudden onset and sudden cessation, was generally centred about the umbilicus, and usually lasted for several minutes. She described it as being colicky in character, and though it was occasionally felt in the back, it did not radiate into either the shoulder or the groin. Vomiting occurred when the attacks of pain were especially severe. The vomit was never excessive, and sometimes contained bile. The appetite was good, and she did not avoid fats. She has taken aperients for years, but did not think that the pain was worse when she was constipated. During the last four months the attacks have been much more frequent and severe. There has been no loss in weight.

ON EXAMINATION.—The patient was admitted to the Royal Infirmary, Liverpool, and a complete X-ray examination was made by Dr. Roberts. He reported that there was a dilated coil of small intestine showing signs of obstruction. The rest of the alimentary tract was normal. From the radiograph the dilated coil seemed to be near the ileocæcal valve, but this appearance was false. It was pelvic in position, but was 6 ft. from the duodenojejunal juncture. The dilated coil was about three times the diameter of the proximal jejunum and at least four times the diameter of the succeeding

bowel. The whole coil was some 10 in. long, and there was a stricture of the bowel at either end of the dilatation, the distal stricture being much narrower than the proximal stricture. The three stercoliths were prominent

FIG. 65.—Inside of dilated coil of jejunum with three enteroliths *in situ*. A, Distal bowel ; B, Stricture ; C. Stricture : D, Proximal bowel.

FIG. 66.—Outside of jejunum. A, Proximal bowel ; B, Distal bowel.

features even from the outside, and they could not be moved from the dilated coil. (*Figs.* 65, 66.)

OPERATION.—This consisted of resection of the coil and its contained concretions. The bowel was restored by closure of both ends and a side-to-side anastomosis. The convalescence was uninterrupted.

PATHOLOGICAL REPORT.—Dr. Howel Evans reports on the concretions as follows :—

" The largest concretion is irregularly pyramidal in shape, the sides measuring approximately 2 × 5 cm. The second is flat and triangular, 2 × 2 × 0·5 cm. The third is similar in shape to the second but somewhat smaller. They are all smooth and polished like pebble stones. They show a thick smooth brown outer layer, and the concretion which was examined could be crushed between the fingers, showing a loose friable centre. The material is largely organic, probably fatty in nature as it is readily soluble in alcohol, leaving a greasy stain when the alcohol is evaporated. The brown colour is chiefly due to urobilin. Tests for unaltered bile-pigments are negative. On extraction with chloroform, cholesterol cannot be demonstrated either by crystallization or chemical tests. Inorganic matter is scanty. Traces of calcium are present, but phosphorus, a frequent constituent of intestinal concretions, cannot be found. Assuming that the two other concretions are similar chemically as they appear to the naked eye, the evidence is against gall-stones and in favour of organic material, largely fats and soaps, which have been formed *in situ*."

A complete analysis of two enteroliths is given in a paper by James Phillips, of Bradford.[1]

REFERENCE.

[1] PHILLIPS, J., *Brit. Jour. Surg.*, 1920–21, viii, 378.

GASTRO-INTESTINAL CRISIS OF ANGIONEUROTIC ŒDEMA.

BY R. J. McNEILL LOVE,
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ABDOMINAL symptoms of angioneurotic œdema have been observed on numerous occasions. Sir William Osler[1] describes a case of a man, age 58 years, who had been affected since childhood with attacks of abdominal colic, retching, and vomiting, two months being the longest interval of freedom. These attacks were associated with patches of œdema, but never of purpura. In the same paper an account is given of a woman of 45 who for five years suffered from attacks of urticaria and œdema of the hands and lips. These manifestations were associated with periods of vomiting which lasted from ten to twenty-four hours. Grey powder and calcium lactate resulted in complete cure. More recently a fatal case of Quincke's œdema with painful abdominal crises has been described.[2]

The case described below is of interest in that radiological abnormalities were evident during the gastric crises, and caused some discussion as to their correct interpretation.

Miss P. B., age 31, stated that she had always been healthy until two years ago. One Saturday evening, while dressing in preparation for a dance,

she found to her surprise that she could see her upper lip. The lip became rapidly swollen. and she was unable to attend the dance. and went to bed instead. On the following day she suffered from abdominal discomfort, but the lip improved, and she returned to work the day after. Since then she has been subjected to sudden œdematous swellings. chiefly of the face, lips, and forearms. and attacks of violent abdominal pain and vomiting. These
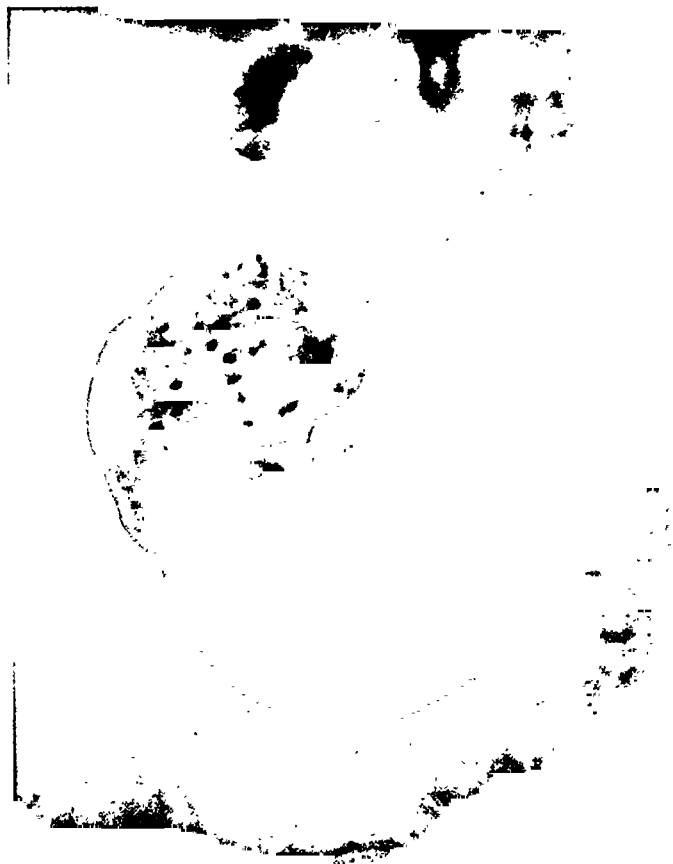
Fig. 67.—Radiograph showing peculiar condition of pyloric portion of stomach, presumably due to œdema.

attacks are ushered in with a feeling of constriction affecting the throat and dysphagia.

A fortnight seldom passes without her being confined to bed for one or two days. Recently these attacks were so severe that she was admitted to the Metropolitan Hospital under Dr. J. W. Linnell. A bismuth meal (*Fig.* 67) revealed a curious condition which was considered to be possibly a polyposis of the pylorus, although the diagnosis of an angioneurotic œdema was held to be more likely. However, as the abdominal symptoms had increased in

severity, and the patient yearned for any possible relief, a laparotomy was performed four days after the radiograph had been taken, in order to exclude any permanent pathological condition. The stomach was practically normal except that the pyloric portion was slightly more red and glistening than usual, and the individual muscular bundles were more apparent. The abdomen was closed, and six days later a radiograph (*Fig.* 68) revealed no abnormality.

Fig. 68.—Radiograph taken ten days later, the stomach now being almost normal.

Three months later (Jan. 12, 1932) the patient reported that although she had had further attacks they were " not so bad and less often ".

As is usual in cases of angioneurotic œdema, the disease in this instance was hereditary. The patient's father and one of his brothers both suffered from the more common subcutaneous manifestations.

REFERENCES.

1 OSLER, SIR WILLIAM, *Brit. Med. Jour.*, 1914, No. 1.
2 VALLERY-RADOT, *Bull. Soc. méd. Hôp. de Paris*, 1931, March 30.

REVIEWS AND NOTICES OF BOOKS.

Tumours of the Breast: their Pathology, Symptoms, Diagnosis and Treatment. By Sir G. Lenthal Cheatle, K.C.B., C.V.O., F.R.C.S., Consulting Surgeon and Emeritus Lecturer on Surgery, King's College Hospital, London ; and Max Cutler, B.Sc., M.D., Director of Tumor Clinic, Michael Reese Hospital, Chicago. Large 8vo. Pp. 596 + viii, with 468 illustrations and 18 coloured plates. 1931. London : Edward Arnold & Co. 50s. net.

This book is the outcome of thirty-five years' study of the breast, and is based on the examination of whole microscopical sections in series which Sir Lenthal Cheatle has developed to such a high degree of perfection. All pathologists and surgeons intimately concerned in the study and treatment of diseases of the breast must have read with interest the numerous papers Sir Lenthal Cheatle has written on these subjects. This volume, in which he has gathered his accumulated material and expanded his theories, will be very welcome to them—none the less because some of the matter it contains is controversial.

The first chapters are devoted to a careful description of the anatomy and physiology of the breast, and the authors have collected in an attractive form all the available knowledge on these subjects. After a survey of the congenital and acquired anomalies of the breast they discuss the natural laws in pathological growth and the relation between hyperplasia and neoplasia.

Perhaps the most important part of the book is that formed by the chapters dealing with the medley of conditions at present included under the heading of 'chronic mastitis'. The authors believe chronic mastitis to be much more a physiological process than a pathological one, and a process of inflammation least of all. To the condition they apply the term 'mazoplasia' and consider it has no relation to carcinoma production. From this they clearly differentiate the condition they call 'cystophorous epithelial hyperplasia', which is one of pathological activity and can be traced in all stages of transition to the neoplastic type, which may consist either of benign or malignant tumours. About 20 per cent of all carcinomas of the breast can be directly traced to this method of origin. They propose to limit the term 'chronic mastitis' to those conditions where definite evidence of the existence of inflammatory changes is forthcoming.

The evidence in support of the authors' suggestions is clearly stated and illustrated. Many observers will, however, feel that the problems of chronic mastitis are too complex to be solved in this simple way, and will therefore prefer to retain the admittedly unsatisfactory term of 'chronic mastitis' until they are convinced that these new terms are not leading to worse confusion. A more authoritative criticism of these views is to be found in the Bradshaw Lecture on chronic mastitis by Sampson Handley (*Lancet*, 1931, Nov. 28).

The sections on cancer of the breast throw new light on the subject, for by their whole-section method the authors have separated a large number of anatomical types not generally differentiated in published series. They do not favour histological grading as a method of prognosis, and indeed it is clear that this method can only be taken into consideration within each distinct anatomical group. As regards the treatment of cancer of the breast, the authors favour radical operation followed by external radiation. Radiation alone is used on those patients who refuse operation or who are unsuitable subjects for operation.

In the chapter on Paget's disease, the various views of its etiology are outlined and an interesting and detailed study of seventeen cases is used as a basis for the interpretation advanced.

In the last chapters, which are devoted to a review of radiation treatment, the action of radium and the factors in radio-resistance are discussed in relation to breast tumours, and the various current views on the efficacy of radiation treatment of cancer of the breast in all stages, and of its metastases, are impartially given. There is much information in these chapters that is indispensable to the general practitioner, to whom patients must always turn for sound advice as to whether radium or surgery is to be used. The book should therefore appeal, not only to the scientific worker, but also to a wider circle of clinicians with whom the ultimate settling of pathological details ranks low in importance.

The volume is arranged so as to be used as a work of reference and as an atlas. It is lavishly provided with illustrations, and both authors and publisher are to be congratulated on the way in which it is presented.

Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London. Third edition, revised and enlarged. Medium 8vo. Pp. 277 + xx, with 318 illustrations, some of which are in colour. 1931. Bristol: John Wright & Sons Ltd. 21s. net.

THE first edition of this work was reviewed in the BRITISH JOURNAL OF SURGERY just after its appearance in 1927. It was then hailed as an admirable book, setting out in a pictorial way a series of demonstrations of the physical signs of clinical surgery, and it was confidently recommended to the notice of all students. Your reviewer can do nothing better than reiterate this notice, and say that the book has maintained the standard which was then set, and has now established itself as a work which every student ought to possess.

At the present time, when the accessory methods of diagnosis are so largely practised and when attempts are made to learn clinical surgery by easy routes, it is more than ever necessary that students should be reminded that nothing can ever take the place of close contact with actual clinical material, the study of which will supply the framework on which all other methods of diagnosis must be based. This book helps very largely to this end, not to supplant such clinical examples, but to assist the student to make the best use of the material which is presented to his notice.

This edition is more liberally illustrated than ever, but a good many of the pictures are really superfluous, and if by a judicious reduction of their number the price of the book could be lowered a little, the student would have every reason to be grateful.

Although the illustrations are nearly all worth framing, there are a few exceptions. For instance, Fig. 80, showing a mastoid abscess, should be replaced by a photograph of an actual case, and Fig. 86, of cavernous sinus thrombosis, is not sufficiently characteristic to convey the main features of that striking condition to the student. Surely it would not be difficult to get a better picture of a backward dislocation of the elbow than the one shown on page 99 ? Figs. 21 and 22 ought to be strictly comparable, and it would add greatly to their value if a picture of gummata on the leg could be substituted for those on the chest. It might be a help for future editions to point out that tumours in muscles, as well as moving with the muscle, are also fixed when the muscle is held in contraction. In dealing with the examination of the thyroid it is rather amusing to have the time-honoured sign associated with the act of swallowing attributed to such a comparative youngster as our friend Lahey. It also seems a pity to omit to mention the valuable aid which we often get from the use of X rays in the elucidation of these swellings.

Fig. 42, illustrating the method of determining the relation of a swelling to the sternomastoid, is a little misleading, for it certainly suggests that the patient is trying to force her head towards the affected side, though the text clearly explains that it should be in the opposite direction. In speaking of thyroglossal and branchial fistulæ, it would be well to mention that the secretion is characteristically sticky. As the text asserts, the risus sardonicus is supposed to resemble a smile, but the smile is surely a painful one, and this is borne out by the wonderful illustration borrowed from John Thomson. In the description of the tests for dislocation of

the shoulder, one wonders why those attributed to Callaway and Duga should be designated by their authors' names, while poor Hamilton is not given credit for his valuable straight-edge test.

In dealing with the examination of the liver for enlargement, it is a mistake to give the impression that inspection is of very little value. Quite often not only can the enlarged liver be seen, but the umbilication of the nodules may be perfectly obvious if the patient is well relaxed and in a good light ; and quite apart from that, the general enlargement of the hepatic region is often very striking. Of course it is possible that the author assumes that such gross conditions will never present a problem in diagnosis, but a considerable clinical experience leads the reviewer to assert that such is unhappily fallacious. In dealing with the umbilicus it would have been well to mention the nodule of malignant growth which is often so helpful in elucidating a doubtful case.

One questions, in these days, whether Trendelenburg's test for varicosity of the internal saphenous is always an indication for the operation associated with his name. Rather it appears as though operations for varicose veins had been entirely superseded by the easier and more satisfactory method of injection.

But these are very minor points, and they do not detract much from what is undoubtedly a very useful and valuable book.

Die Schleimhaut des Verdauungskanals im Röntgenbild. By HENRI CHAOUL and ALBERT ADAM, with a Foreword by FERDINAND SAUERBRUCH. Imperial 8vo. Pp. 229 + viii, with 219 illustrations. 1931. Berlin and Vienna : Urban & Schwarzenberg. Paper covers, RM. 22.50 ; bound, RM. 25.

THIS volume is a record of work in a field which has hitherto attracted more attention among Continental radiologists than in this country. The critical radiographic study of details of the alimentary mucosa which received its initial impulse from the work of Forsell, has been advanced by many workers. In this field Chaoul, himself a pioneer, has developed a technique and special apparatus which facilitate and lend precision to this somewhat laborious and time-consuming work, which consists essentially in radiographing the organs when outlined by *very small quantities* of opaque emulsion, under suitable compression.

A fundamental fact which emerges from the work under review is the constancy of the arrangement of the gastric mucosal pattern in the same individual, and to some extent in different individuals. This is at variance with the views of Forsell and of Berg, but the authors appear to have proved their case. They discuss fully the influence of tone and peristalsis on the mucosal pattern, and ascribe to these factors, rather than to independent contraction of the muscularis mucosæ (Forsell), the predominant rôle in producing its momentary variations.

In many respects their study of the normal is the most valuable part of the book. On the pathological side there is little which is new, though the standard of technical achievement compels admiration, and many excellently reproduced radiograms of small lesions, which might easily have escaped recognition by ordinary methods, prove the value of the authors' technique; for example, peptic ulcers of the œsophagus, early carcinomata of the stomach, gastrojejunal ulcers, and a striking case of papillomata of the colon.

We had hoped that their researches might throw new light upon the diagnosis of gastritis and colitis, and particularly upon the causation of 'mottling' of the mucosa which, in a radiogram, often suggests these conditions. These appearances are discussed, and such causative factors as mucosal hypertrophy and atrophy, papilliform swelling, and deposits of mucus are differentiated, but it is clear that the accurate radiological diagnosis of gastritis and colitis requires further study, both radiologically and pathologically : in the meantime we must endorse the rather cautious views taken by the authors towards these problems.

The book may be heartily recommended to gastro-enterologists and to radiologists, who will find it both informative and stimulating. There is a very complete bibliography.

Lehrbuch der Röntgendiagnostik. By H. R. Schinz, W. Baensch, and E. Friedl, with the collaboration of M. Holzmann, A. Hotz, O. Jüngling, E. Liebmann, E. Looser, and K. Ulrich. Third edition. Two volumes. Imperial 8vo. Pp. 1623 + xxviii, with 2714 illustrations and 5 photographic plates. 1932. Leipzig: Georg Thieme. Paper covers, M. 214; bound, M. 220.

This comprehensive text-book of radiology has won a well-merited international reputation. The third edition represents a very distinct advance upon the earlier ones, both in the wealth of additional matter and in the elaboration of the original sections, which have been largely rewritten. One of the most valuable features of this book has been the authors' insistence upon the pathological basis of X-ray appearances : this aspect is even more fully emphasized in the new edition. For example, in the section on bone growth and diseases of bone are collected illustrations of normal and growing bones with clear descriptions of normal growth processes. Diseases of bone are dealt with on similar lines, with photographs of specimens, microphotographs, and key diagrams side by side with the radiograms. This section contains also descriptions of rare bone diseases and of conditions which have been only recently studied in detail by X rays. In particular the chapters on diseases of the vertebral column have been rewritten in the light of the revolutionary work of Schmorl.

In compiling this work the authors owe much to their collaborators (Holzmann, Hotz, Jüngling, Liebmann, Looser, and Ulrich). In addition, the best recent work in all countries has been laid under contribution, including that of the American registry of bone sarcoma ; German work on tuberculosis of the lungs, Scandinavian and German work on the alimentary mucosa, and French work on cardiology. The chapter on radiology in gynæcology is new, and that dealing with urology has been brought up to date by inclusion of new methods in urography. The excellent articles by Jüngling on myelography and ventriculography remain as originally written.

About half the second volume is devoted to the chest, and half to the alimentary tract. The sections dealing with pulmonary tuberculosis and with radiology in connection with thoracic surgery are specially useful. The alimentary tract is extremely well done, with emphasis throughout upon 'direct' as opposed to indirect signs. A full index and really useful bibliographies—the latter placed where they are most useful, at the end of each short section—are good features.

Very high praise is due to the publishers for their share in this work ; particularly for the radiograms, which reproduce the crispness and clarity of the originals. Their wide experience of this type of work, and their immense material, have enabled them to publish this book supremely well ; and it is good to find collected within its covers material which has already appeared in periodical form, but which in that form is difficult of access. This book is the best and most complete modern text-book on diagnostic radiology that exists, and it should be in daily use by every radiologist and on the shelves of every important medical library.

Atlas de Radiographie osseuse.
  I. Squelette normal. By G. Haret, A. Dariaux, Electro-radiologistes des Hôpitaux de Paris ; and Jean Quénu, Professeur agrégé à la Faculté de Médecine, Chirurgien des Hôpitaux de Paris. With the collaboration of H. P. Chatellier, Oto-rhino-laryngologiste des Hôpitaux. Second edition, revised and enlarged. Preface by Pierre Duval. Large 4to. Pp. 186, with 149 illustrations. 1932. Paris : Masson et Cie. Fr. 200.

  II. Pathologie. In two volumes. Vol. I. Lésions traumatiques. By G. Haret and A. Dariaux, Electro-radiologistes des Hôpitaux de Paris ; and Jean Quénu, Professeur agrégé à la Faculté de Médecine, Chirurgien des Hôpitaux de Paris. Vol. II. Lésions non traumatiques. By Etienne Sorrel, Chirurgien de l'Hôpital Trousseau ; and Mme. Y. Sorrel-Dejerine, Ancien Interne des Hôpitaux. Large 4to. Pp. 344, with 897 illustrations. 1931. Paris : Masson et Cie. In two volumes, Fr. 310 ; in one volume, Fr. 280.

It is difficult to do justice to this magnificent work, the first part of which has already entered upon a second edition. The first part gives a most complete radiographic presentation of the normal skeleton. First, the bones of an adult, each from

several different points of view. Second, the bones of a child at various ages from intra-uterine life up to puberty. Third, the various possible supernumerary bones. Every radiogram is beautifully reproduced and accompanied by an outline diagram in which all the special points are emphasized.

The second part, dealing with abnormal conditions, is in two volumes, the first being concerned with fractures and other injuries. Those regions specially prone to injury are most profusely illustrated : thus there are no fewer than twenty-nine radiograms of the fractures in or near the wrist, and every one of these is accompanied by a very clear outline diagram. For the same reason all the injuries near the large joints are generously illustrated. Fractures and dislocations of the spine are well figured. The figures of the fractures of the skull might well be increased in number and variety. There is a short section on gunshot injuries of bones and a few illustrations showing the different density of callus at periods varying from ten to eighty days. We hope the next edition will contain more figures showing varieties of delayed union, non-union, and pseudarthrosis.

The second volume of the second part illustrates the various diseases of the bones and joints. We confess that we have been a little disappointed in this because it does not approach the first two parts (normal skeleton and traumatic lesions) in completeness. Perhaps this is inevitable because the range of the subject is so much greater in the matter of diseased conditions. The section on tuberculosis of the bones and joints, forming nearly half the volume, is good and the figures are well chosen and typical. Syphilis is only scantily illustrated. New growths are shown in seventeen figures, which are quite inadequate for the subject. The rest of the book treats of various conditions, osteomyelitis, arthritis, dystrophies, and congenital malformations, and is full of good pictures of these conditions.

The conception of this book is very good, and it will form an invaluable work of reference. We foresee that the authors will soon have an opportunity of enlarging the section dealing with diseased conditions on the lines we have indicated.

Röntgenuntersuchung und Strahlenbehandlung der Speiseröhre. By Dr. JOSEF PALUGYAY (Vienna). Handbuch der Röntgenkunde, Vol. III. Royal 8vo. Pp. 392 + xiv, with 224 illustrations. 1931. Vienna : Julius Springer. Paper covers, RM. 56 ; bound, RM. 59.60.

THIS book bears the hall-mark of the publishing house of Julius Springer of Vienna. The author's main object has been to study by radiology the œsophagus in health and disease. Experiments have been performed by radiological means with the subject in the horizontal position and with the pelvis raised ; the knowledge obtained has been applied to the physiology and pathology of the œsophagus. One chapter is devoted to the technique of this research. The author has not confined himself to radiology, but has presented a consideration of the pathology, symptoms and signs, differential diagnosis, and treatment of the various abnormalities of the œsophagus. The book is arranged in an orderly manner, and opens with an historical section. Some of the most important facts are re-stated in connection with the anatomy and physiology of the œsophagus. Attention is drawn to the variations in length of the œsophagus in different individuals and in the two sexes : greater variations are stated to occur in width. Interesting stereographic diagrams are given of transverse sections of the œsophagus in different regions with inspiration and expiration. In view of the present-day prominence of the sympathetic nervous system, the section on the nervous connections of the œsophagus is disappointing. The physiology of deglutition is considered fully and all theories are examined ; the author reaches the conclusion that the act is very complicated, involving the co-ordination of the musculature of the pharynx, œsophagus, cardia, and diaphragm, and he disagrees with Magendie's division into three parts. Careful study has been made of deglutition with the body horizontal and also with the pelvis raised, and interesting conclusions have been reached.

Functional diseases of the œsophagus, with special regard to the radiological appearances, receive due attention, and no detail, however small, escapes the author's

attention, which is merited when we consider our ignorance of these subjects. The subject of cardiospasm is dealt with comprehensively with due regard to a consideration of the pathology. No statement, however, is made on the possible involvement of the sympathetic nervous system in this condition. A very useful description of the many varied radiological appearances in this condition is given, and the author states that it is possible to estimate the thickness of the cardia by radiography.

Organic diseases occupy a large portion of the book. An interesting account of diverticula is given—pulsion diverticula being divided into pharyngo-œsophageal (Zenker's) and œsophageal pulsion diverticula, which may occur anywhere in the œsophagus, according to the author. He states that there are two varieties of traction diverticula—first, there is the type produced by traction outside the wall plus pressure from within the lumen ; secondly, the rudimentary type caused by adhesions of the œsophageal wall to its environment, hindering contraction of the wall.

Many clear radiological pictures of carcinoma of the œsophagus are given, and we are glad to see the statement made that carcinoma occurs in all parts of the œsophagus, which agrees with our experience. A section is devoted to œsophageal operations, together with a full consideration of the possibilities of the treatment by radium of carcinoma. The author states that carcinoma of the œsophagus in any other position than the cervical can only be treated by radium or X rays. The indications, contra-indications, and complications are discussed.

The whole work merits high praise. There is a good bibliography of the literature, the diagrams are clear, and the radiological pictures excellent.

Röntgendiagnostik der Gallenblase. By Priv.-Doz. Dr. F. EISLER and Dr. G. KOPSTEIN. Radiologische praktika, Bd. XVII. Super royal 8vo. Pp. 153 + viii, with 151 illustrations. 1931. Leipzig : Georg Thieme. Bound, RM. 18.60.

THIS book keeps up the high standard already achieved in this series of works on radiology. The authors give a description of the anatomy and physiology of the biliary tract, pointing out how cholecystography has been of value in this sphere as well as in diagnosis. The technique of the oral and intravenous methods of cholecystography are described in detail. The radiological technique occupies very little space, and rightly so, the book being written from the point of view of interpretation of the radiograms, and correlation between the X-ray and clinical or operative findings. The reproductions of the numerous and excellent radiograms are good, the conditions referred to showing up well. There is a useful bibliography of over 350 references, which is an adequate summary of the growing literature on the subject.

Annals of Roentgenology: a Series of Monographic Atlases. Edited by JAMES T. CASE, M.D., Professor of Roentgenology, North-western University Medical College, Chicago. Volume VII, Urological Roentgenology. By HUGH H. YOUNG, M.D., Clinical Professor of Urology, Johns Hopkins University ; and CHARLES A. WATERS, M.D., Associate in Clinical Roentgenology, Johns Hopkins University. Second edition revised. Large 4to. Pp. 564 + xlix, with 592 illustrations. 1931. New York : Paul B. Hoeber Inc. $20.00.

THIS is a very large book of some 564 pages ; it is most handsomely printed and bound, and contains no fewer than 592 illustrations. In it will be found an enormous amount of information on almost every aspect of the surgery of the genito-urinary tract ; it is by no means a mere atlas of skiagrams, for the text includes articles on the pathology, the diagnosis, and the treatment of the varied lesions of this tract. The authors' plan has been to discuss such subjects as tuberculosis, stone, and neoplasms of the genito-urinary tract and to illustrate them by skiagrams and drawings ; accompanying these the case histories are invariably given.

Perhaps the most interesting of the chapters is that on arteriography of the aorta and its abdominal branches ; for the purpose of showing the circulation of the kidneys 20 c.c. of 100 per cent solution of sodium iodide are injected into the aorta in the region of the first lumbar vertebra ; apparently this can be done without.

.much risk, for the middle coat of the aorta produces prompt occlusion of the puncture. The resulting skiagrams are very striking, and show, for instance. the few vessels present in a tuberculous kidney as compared with those of the opposite organ. The authors sum up their present attitude to this novel method of investigation : " It is needless to say that one hesitates to express an opinion at this time upon this startling work by dos Santos and his confrères. Coming as it does from a man of marked scientific attainments. and with the assurance that puncture of the aorta has been done in 150 cases without untoward results. one must maintain an open mind. express the greatest interest in this remarkable presentation, and hope that future experience will bear out the conclusions of the authors."

We note that the authors have tried pyeloscopy and have abandoned it in favour of serial pyelograms ; they consider that this method of investigation has little to add in the way of information and that it has some very distinct disadvantages. There is an excellent chapter on neurological lesions involving the urinary tract.

The book has one drawback. its rather high price, but we can recommend it as a real contribution to the surgery of the genito-urinary tract ; it will, we think, be used chiefly as a book of reference.

On page 132. two skiagrams have been inserted upside down, but apart from this there are singularly few mistakes or misprints.

A Descriptive Atlas of Radiographs: An Aid to Modern Clinical Methods. By A. P. BERTWISTLE, M.B., Ch.B.. F.R.C.S.E. Second edition, revised and enlarged. Crown 4to. Pp. 552 + xxviii, with 767 illustrations. 1932. London : Henry Kimpton. 42s. net.

THE author is to be congratulated on his energy and diligence in bringing together this large collection of radiographs and on the fact that a second edition of the book has already been called for. The originals of most of the pictures have been supplied by various workers in special departments—Sir William Milligan, the nasal sinuses ; Mr. Marxer, the dental ; Professor Wilkie and Dr. Spriggs, the alimentary ; Professor Fullerton and Mr. Kidd, the urinary ; Professor Sicard and Mr. Dott, the nervous ; and Mr. Morriston Davies, the chest.

The book constitutes a veritable storehouse of information and illustration. Our chief criticism is that the reproduction of the more complicated radiograms— those of the chest and abdomen, for example—is not clear enough to make these pictures of much value for the purpose of reference.

The Rational Treatment of Varicose Veins and Varicocele. By W. TURNER WARWICK, M.A., M.B. (Cantab.), F.R.C.S., Surgeon to Outpatients and Surgeon in charge of the Rectal Department, the Middlesex Hospital, London. Crown 8vo. Pp. 188, with 12 illustrations. 1931. London : Faber & Faber. 5s. net.

IN addition to the treatment of varicose veins this book also gives a very exhaustive account of the anatomy and physiology of the veins of the lower extremity. The chapter on the etiology of varicose veins, of no fewer than 43 pages, deals most clearly and in great detail with the subject from the historical, experimental, and practical sides and is quite the best account of the subject we have seen. Although he describes the operations for varicose veins, the author considers them replaced by the injection treatment, which is described in detail.

The pathology and etiology of varicocele, as well as its treatment both by operation and injection, are described.

The author also gives an account of the injection treatment of piles. For first and second degree piles injection is the method of choice, and in those of the third degree it is worthy of a trial if, for any reason, operation is contra-indicated. The use of a 20 per cent solution of phenol in equal parts of glycerin and water is the solution preferred, and the injection of the solution into the base of the pile is emphasized.

To all who want a clear and simple account of the treatment of the three common conditions mentioned above, as well as those requiring a more academic account of varicose veins, this book should give all that is required.

Surgery. By S. K. SEN, L.R.C.S., L.R.C.P. (Edin.), L.R.F.P.S. (Glas.), late State Surgeon, Nepal. Vol. III. Surgery and Pathology of Growths. Demy 8vo. Pp. 348 + viii, with 56 plates. 1931. Calcutta and Burdwan : The Surgical Education Society Ltd., Rs. 7/8 per vol.

" THIS is the third of the series of eight volumes of Surgery, the whole plan of which is described in the preface of the first volume. It contains the description of all pathological Regenerations and New Formations. The book is intended for those mid-senior students who have just finished a course of Pathology, and are attending Surgery classes.

" It is written to the standard of qualifying examinations in England. Being a student of Edinburgh I have mainly followed the teachings of the Edinburgh School of Medicine where I attend the ordinary classes even now when opportunity offers itself. In fact many pages are written from the lecture notes taken by me in the University classes very recently."

These first two paragraphs of the Preface explain clearly the intention of the author. The book is fairly well illustrated with plates made from the author's own cases. It is quite likely that a book written in this way will be more useful to the Indian student than the average European text-book.

Minor Surgery. By LIONEL R. FIFIELD, Late Surgical First Assistant and Registrar, London Hospital. Second edition, revised by R. J. McNEILL LOVE, M.S. (Lond.), F.R.C.S., Surgeon, the Royal Northern Hospital, etc. Crown 8vo. Pp. 440 + viii, with 281 illustrations. 1931. London. H. K. Lewis & Co. Ltd. 12s. 6d. net.

THE second edition of this well-known handbook contains few alterations or additions. It is a thoroughly sound book to give to a student beginning surgery, containing, as it does, such often neglected topics as how to pass a catheter, apply a plaster, or extract a tooth. The use of tannic acid for burns, the injection treatment of varicose veins, and the methods of treating minor affections of the ear, are welcome additions. The author will not receive a general acceptance of his theory that Volkmann's contracture is " caused by an extravasation of blood within the sheaths of the flexor muscles ". If any serious criticism must be made, it is that many methods of treatment are given without sufficient stress upon the principles which should govern their selection.

Surgical Pathology of Prostatic Obstructions. By ALEXANDER RANDALL, M.A., M.D., Professor of Urology, University of Pennyslvania. Large 8vo. Pp. 266 + xiii, with 78 plates. 1931. London : Baillière, Tindall & Cox. 37s. 6d. net.

THIS monograph is an important contribution to the literature of urology ; it is the result of ten years' study of the autopsies at the Philadelphia General Hospital, an institution with over two thousand beds and a mortuary in which two or more autopsies are performed daily. During this period 1218 specimens of the male bladder and prostate were examined, and later their case histories were obtained and correlated. Hardly any of these cases of prostatic obstruction had been operated on, and this is explained by the fact that the Philadelphia General Hospital, in addition to the usual medical and surgical divisions, comprises departments for tuberculous and insane patients and " also functions as the city almshouse and home for the indigent ".

The author describes and illustrates the gross morbid pathology of the various affections of the prostate—hypertrophy, median bar, carcinoma, and abscess—and he discusses the surgical handling of the obstruction produced by such lesions. There are numerous plates showing the various morbid conditions of the prostate ; they are all photographic, and, whilst perhaps they are as good as modern photography can produce, some of them fail to satisfy the reader, and they leave a feeling that Professor Randall would have been better advised to have given fewer plates and to have had them drawn by a competent artist.

One point of interest is that the negro population forms no less than 30 per cent

of the admissions to this great hospital ; they not only suffer from prostatic obstruc-
tion but they suffer from it at an earlier age than the whites ; this confirms what
Freyer affirmed as the result of his experience in India, that the black races are
really ten years older than their age as compared with the white.

As the outcome of his studies of benign hypertrophy, the author divides pros-
tatic enlargement into no fewer than eight varieties : we consider this subdivision
unnecessary, for almost certainly they are merely stages in the development of this
disease. From an examination of seventeen specimens of carcinoma of the prostate
he concludes that there is no foundation for the statement made by Tandler and
Zuckerkandl that this disease starts usually in the posterior lobe ; and again, though
thirteen out of the seventeen had metastases, there were only four instances of
secondary deposits in bone.

Altogether this book is well worth reading by those who are interested in the
surgery of the prostate.

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Surgical Pathology of the Diseases of Bones. By ARTHUR E. HERTZLER, M.D., Profes-
sor of Surgery, University of Kansas. Hertzler's Monographs on Surgical Pathology.
Royal 8vo. Pp. 272 + xiv, with 211 illustrations. 1930. London : J. B. Lippincott
Co. 21s. net.

THIS is one of a series of monographs by Dr. Hertzler on special surgical pathological
subjects. He gives a clear description of all bone diseases, with a good bibliography
of the classical articles on each subject at the end of every chapter. On the many
points of controversy in bone pathology the author does not, in most cases, express
his opinion but gives a review of those held by other authorities. For tumours he
wisely adopts the classification of the American registry of bone tumours. The
chapter on diseases of unknown origin, dealing with such conditions as Perthes',
Köhler's, Schlatter's diseases, etc., and fragilitas ossium, gives us no new help ; and
indeed no reference is made to calcium metabolism or lesions of the parathyroid
glands in the description of osteitis fibrosa cystica. The illustrations are mostly
very good, but some of the skiagrams are not of very typical specimens ; for example,
there is not shown a really typical skiagram of a benign giant-celled tumour.

This book should be useful for those who want to obtain a broad knowledge of
the pathology of bone diseases, but for those requiring special knowledge on some
particular condition there is hardly enough detail given.

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The Laboratory in Surgical Practice. By C. E. DODDS, M.V.O., M.D., Courtauld Professor
of Biochemistry in the University of London, etc. ; and LIONEL E. H. WHITBY, C.V.O.,
M.D. (Camb.), M.R.C.P. (Lond.), D.P.H., Bacteriologist to the Middlesex Hospital,
Bland-Sutton Institute of Pathology, etc. Demy 8vo. Pp. 187 + x. Illustrated.
1931. London : Constable & Co. 8s. 6d. net.

THIS is the first of a new series of surgical monographs under the general editorship
of Gordon Taylor. It describes in clear and simple fashion the numerous laboratory
tests which are employed in surgical work, and includes such clinical methods as
intravenous pyelography. It will be useful to the actual laboratory worker, but
should be read by surgeons who call for these tests. The large experience of the
authors in such investigations is an assurance of the reliability of their statements
as to what can and what cannot be expected from them. The book is well printed,
easy to read, and has a number of useful diagrams.

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Schmerzverhütung : Zwölf Vorlesungen. By Dr. FRITZ STARLINGER (Vienna). Medium
8vo. Pp. 106 + vi. 1931. Vienna : Julius Springer. RM. 6.60.

THIS is a small book, divided into twelve chapters, on various methods of the preven-
tion of pain, by the Assistant in the Eiselsberg Clinic of the University of Vienna. It
is written in German for the use of students and young doctors, and contains articles
on various forms of ether narcotics and forms of anæsthesia.

Guy's Hospital Reports. Edited by ARTHUR F. HURST, M.D. Vol. LXXXI (Vol. XI, Fourth Series). No. 3. July, 1931. Royal 8vo. Pp. 253–378. Illustrated. 1931. London: The Lancet Ltd. Single numbers, 12s. 6d. net; annual subscription, £2 2s. net.

THIS volume contains a description of a study of osteitis fibrosa cystica associated with tumour of the parathyroid, and a detailed report of a case is given. The size of the tumour was 2·7 by 2·2 by 2 cm., and the outstanding feature of the case was hypercalcæmia. In the same volume there is another short article on the condition of the teeth of this patient. It appears that the growth of the teeth ceased soon after the age of 15 years, or became much retarded, the result being analogous to the arrested growth of the long bones. There is a remarkable contrast between the rarefaction of the bones and the comparative integrity of the teeth.

Specific Changes in the Blood Serum. A Contribution to the Serological Diagnosis of Cancer and Tuberculosis. By S. G. T. BENDIEN, Serological Laboratory, Zeist, Holland. Translated by A. PINEY, M.D., Director of the Pathological Department, the Cancer Hospital, London. Royal 8vo. Pp. 95 + xii, with 64 illustrations and 5 spectra in the text and on 8 plates. 1931. London: Wm. Heinemann (Medical Books) Ltd. 10s. 6d. net.

THIS is a very excellent translation of a book which contains a great deal of highly technical material and is therefore of greater interest to the serologist than to the clinician, who, at the present stage, must be largely guided by the findings of other laboratory workers in their attempts accurately to evaluate the author's various claims and theories in connection with cancer. It cannot, in fact, be said that this particular book makes these theories entirely clear, but as Dr. Bendien explains in his preface that he is only at the beginning of the way and that his conclusions are probably not absolutely correct, his book must be read and judged with these facts in view. In any case he gives the necessary details with the aid of which others may carry out the flocculation test, which, it is claimed, may give guidance in cases of tuberculosis and carcinoma. How much practical assistance to clinicians will be forthcoming must be decided by the extent to which other investigators are able to confirm the author's experimental results, particularly in their relation to clinical conditions.

L'Ostéosynthèse des Os longs. Etude critique, biologique et pratique. By C. E. CORNIOLEY (Geneva). Preface by Prof. CH. JULLIARD. Royal 8vo. Pp. 422 + xvi, with 102 illustrations in the text and 16 plates. 1931. Paris: G. Doin et Cie. Fr. 75.

THIS monograph represents a very fair and able description of the author's experience in the operative treatment of fractures, gained in twenty-six years' experience. A very great number of different methods of fixation of the bones are described, and some of these are illustrated by means of line diagrams; but neither the descriptions nor the illustrations would suffice to enable the method to be carried out by these instructions. The reaction of the living bone towards metallic suture material is well illustrated, but the fundamental necessity of mechanical efficiency in fixation is not made equally clear. Throughout the whole book, and especially in the chapter on forearm fractures, there are many cases quoted and illustrated in which the metallic fixation is obviously inadequate to secure immobilization. The osteoperiosteal graft is mentioned, and the cases figured in which it has been used serve only to prove its inefficiency. It is sad to see the operative treatment of fractured neck of the femur only illustrated by the long carpenter's screw method.

The book does not profess to describe other methods of fracture treatment besides those of open operation, but a certain number of splints are mentioned and their uses discussed. The very great importance of skeletal traction and its use as an indispensable preliminary to open operation is not given the place it deserves.

BOOK NOTICES.

*[The Editorial Committee acknowledge with thanks the receipt of the following
volumes. A selection will be made from these for review, precedence being
given to new books and to those having the greatest interest for our readers.]*

United States Army X-ray Manual. Authorized by the Surgeon-General of the Army.
Second edition, rewritten and edited by Lieut.-Colonel H. C. PILLSBURY, M.D., U.S.A.
Crown 8vo. Pp. 482 + xvii, with 228 illustrations. 1932. London : H. K. Lewis
& Co. Ltd. 25s. net.

Ikonographia urologica. Edited by ALFRED ROTHSCHILD (Berlin) ; VICTOR BLUM
(Vienna) ; FRIEDRICH NECKER (Vienna). Part IV. Pp. 21, with 25 illustrations.
1932. Berlin : Georg Stilke. RM. 5.40.

The Technique of the Non-padded Plaster Cast. By FRITZ SCHNEK, M.D. (Vienna).
With a Preface by LORENZ BÖHLER, M.D. (Vienna). Translated by DOUGLAS D.
TOFFELMIER, M.D. (Oaklands, California). Royal 8vo. Pp. 140 + viii, with 169
illustrations. 1932. Vienna : Wilhelm Maudrich. $5 post free.

Technique de l'Ostéosynthèse. By ROBERT DANIS, Professeur de Clinique chirurgicale
à l'Université de Bruxelles. Royal 8vo. Pp. 162, with 149 illustrations. 1932.
Paris : Masson et Cie. Fr. 55.

Konstitution und Vererbung in der Orthopädie. By Prof. Dr. B. VALENTIN (Hannover-
Kleefeld). Royal 8vo. Pp. 30, with 20 illustrations. 1932. Stuttgart : Ferdinand
Enke. RM. 2.50.

Der heutige Stand der Knochenbruchbehandlung. By MORITZ BORCHARDT. Hefte
zur Unfallheilkunde, Heft 11. Royal 8vo. Pp. 72, with 30 illustrations. 1932.
Berlin : F. C. W. Vogel. RM. 7.80.

Handbuch der Unfallmedizin. By Dr. CONSTANTIN KAUFMANN (Zürich). Vol. I.
Allgemeiner Teil. Unfallverletzungen. Royal 8vo. Pp. 862 + xxviii. 1932. Stuttgart :
Ferdinand Enke. Paper covers, RM. 52 ; bound, RM. 55.

Tuberculose osseuse et ostéo-articulaire. By ETIENNE SORREL, Chirurgien de
l'Hôpital Trousseau, and Mme. SORREL-DEJERINE, Ancien Interne des Hôpitaux de
Paris. Royal 4to. Pp. 514, with 640 illustrations. 1932. Paris : Masson et Cie.
Fr. 350.

Surgical Pathology of the Female Genital Organs. By ARTHUR E. HERTZLER, M.D.,
Professor of Surgery, University of Kansas. Hertzler's Monographs on Surgical
Pathology. Royal 8vo. Pp. 346 + xxii, with 285 illustrations. 1932. London :
J. B. Lippincott Co. 21s. net.

Tratado de Patología quirúrgica general. By Dr. MANUEL BASTOS ANSART (Madrid).
Large 8vo. Pp. 853 + xx, with 473 illustrations. 1932. Barcelona : Editorial Labor,
S.A. No price given.

Spezielle Chirurgie der Gehirnkrankheiten. Edited by Prof. FEDOR KRAUSE. Vol.
II/2, Die epileptischen Erkrankungen, by Prof. FEDOR KRAUSE and Dr. HEINRICH
SCHUM. Royal 8vo. Pp. 523–957 + xx, with 95 illustrations. 1932. Stuttgart :
Ferdinand Enke. Paper covers, RM. 63 ; bound, RM. 65.50.

Baillière's Synthetic Anatomy. A Series of Drawings on Transparent Sheets for
facilitating the Reconstruction of Mental Pictures of the Human Body. By J. E.
CHEESMAN, Deputy Medical Officer of Health for Leyton. London. Parts I–XII
and binding case 7½ × 9 in. 156 plates. 1932. London : Baillière, Tindall & Cox.
42s. net.

Leitfaden der Kosmetik für die ärztliche Praxis. By Prof. Dr. A. BUSCHKE. Dr.
ALFRED JOSEPH, and Dr. WERNER BIRKENFELD (Berlin). Large 8vo. Pp. 224 + iv,
with 64 illustrations. 1932. Berlin and Leipzig : Walter de Gruyter & Co.

Some Radium Cases at the Middlesex Hospital: A Photographic Record. By A. CAMERON MACLEOD, M.B., B.S. (Lond.), F.R.C.S., Late Surgical Registrar, Middlesex Hospital. *Large 8vo.* Pp. *154 + viii, with 122 illustrations.* 1931. London: John Murray. 7s. 6d. net.

Allgemeine und spezielle Elektrochirurgie. By Dr. Med. HANS VON SEEMEN (Münich). Royal 8vo. Pp. 474 + xii, with 347 illustrations. 1932. Berlin: Julius Springer. Paper covers, RM. 62; bound, RM. 66.80.

Die Formfehler und die plastischen Operationen der weiblichen Brust. By Dr. ERNA GLÄSMER, Frauenärztin in Heidelberg. Royal 8vo. Pp. 94, with 48 illustrations. 1930. Stuttgart: Ferdinand Enke. Paper covers, RM. 6.30; bound, RM. 7.65.

Arthritis deformans und chronische Gelenkkrankheiten. By Prof. Dr. HANS BURCKHARDT (Essen). Royal 8vo. Pp. 464 + xii, with 70 illustrations. 1932. Stuttgart: Ferdinand Enke. Paper covers, RM. 53; bound, RM. 55.50.

Die unspezifischen chronischen Erkrankungen der Wirbelsäule. By Prof. Dr. HANS BURCKHARDT (Essen). Royal 8vo. Pp. 77 + vi, with 22 illustrations. 1932. Stuttgart: Ferdinand Enke. RM. 7.50.

Plastic Surgery of the Nose, Ear, and Face. By Dr. VICTOR FRÜHWALD, Ex-assistant of the Nose, Throat and Ear Clinic, Vienna. Translated by GEOFFREY MOREY, M.B., B.S. (Adelaide), D.L.O. (Lond.). Large 8vo. Pp. 86 + viii, with 88 illustrations. 1932. Vienna: Wilhelm Maudrich. $4 post free.

A Short Practice of Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, etc.; and R. J. McNEILL LOVE, M.S. (Lond.), F.R.C.S., Surgeon, Royal Northern and Metropolitan Hospitals, etc. Demy 8vo. Vol. I. Pp. 536 + viii, with 269 illustrations. 1932. London: H. K. Lewis & Co. Ltd. 20s. net.

Orthopædic Surgery. By WALTER MERCER, M.B., Ch.B., F.R.C.S.E., F.R.S.E., Assistant Surgeon, Royal Infirmary, Edinburgh, etc. With a Foreword by JOHN FRASER, M.C., M.D., M.Ch., F.R.C.S.E., Regius Professor of Clinical Surgery in the University of Edinburgh. Medium 8vo. Pp. 695 + xii, with 371 illustrations. 1932. London: Edward Arnold & Co. 32s. 6d. net.

An Introduction to 'Avertin' Rectal Anæsthesia. By J. KEMPSON MADDOX, M.D., Ch.M. (Sydney), M.R.C.P. (Lond.), Tutor in Medicine, University of Sydney. With a Foreword by HAROLD R. DEW, M.B., F.R.C.S., F.R.A.C.S., Professor of Surgery, University of Sydney. Large 8vo. Pp. 124. Illustrated. 1931. Sydney: Angus & Robertson. 9s. net.

Edemi chirurgici. By Dott. OSVALDO AMOROSI. Medium 8vo. Pp. 228 + vi, with 28 illustrations. 1932. Lanciano: Giuseppe Carabba.

Le Ascaridiosi chirurgiche. By Dott. ETTORE RUGGIERI. Medium 8vo. Pp. 256 + vi, with 29 illustrations. 1932. Lanciano: Giuseppe Carabba.

Principles of Preoperative and Postoperative Treatment. By REGINALD ALEX CUTTING, M.D., C.M., M.A., Ph.D., Assistant Professor of Surgery, Louisiana State University Medical Centre. With a Foreword by RUDOLPH MATAS (New Orleans). Super royal 8vo. Pp. 812 + xx, with 76 illustrations. 1932. New York: Paul B. Hoeber, Inc. $10.00.

The Use of Lipiodol in Diagnosis and Treatment. By J. A. SICARD, Late Professor in the Faculty of Medicine, Paris, and J. FORESTIER (Aix-les-Bains). Royal 8vo. Pp. 235 + x, with 50 illustrations. 1932. London: Humphrey Milford, Oxford University Press. 16s. net.

Intracranial Tumours. By HARVEY CUSHING, Professor of Surgery, Harvard Medical School. Super royal 8vo. Pp. 150 + xii, with 111 illustrations. 1932. London: Baillière, Tindall & Cox. 26s. 6d. net.

Primary Carcinoma of the Lung: Branchiogenic Cancer: A Clinical and Pathological Study. By B. M. FRIED, M.D., Peter Bent Brigham Hospital, Boston, Mass. Royal 8vo. Pp. 247 + x, with 95 illustrations. 1932. London: Baillière, Tindall & Cox. 26s. 6d. net.

SOME BYGONE OPERATIONS IN SURGERY.

BY SIR D'ARCY POWER, K.B.E., LONDON.

X. A CASE OF STRANGULATED UMBILICAL HERNIA. QUEEN CAROLINE OF ANSPACH.

(Concluded.)

" ON Sunday morning about nine o'clock, the surgeons upon opening the Queen's wound found the mortification was not spread ; and upon cutting off what was already mortified, declared that she might recover. This appeared so inconsistent with their declarations some few hours before, and in my opinion showed so much ignorance. that if a life of this consequence committed to the care of four of the best physicians and three of the best surgeons in England received no better assistance from their skill, how natural it is to deplore the situation of those whose safety depends on the sagacity of these professions and how reasonable to despise those who put their trust in such aids. Not that I am so unjust to surgery as to put that science upon the same foot with physic ; and, for my own part, I firmly believe that there was not the least mortification begun, when they ignorantly pronounced there was ; and that what they cut off was not mortified and only declared so to conceal the mistake they had made the night before in saying it was.

" Monday and Tuesday the Queen was what the doctors, surgeons and courtiers called better, there being no threatening symptoms in her wound and her vomitings being much slackened ; but nothing passing through her those who judged by essential circumstances. and not on the hourly variation of trifles whatever they might say from fashion or to please, could not in reality believe the Queen's condition more hopeful or less dangerous whilst that main point of the internal stoppage continued in the same situation ; and whenever the King used to tell her how much better the doctors and surgeons said she was and the hope they gave him, the moment his back was turned she used to look at the Princesses, shake her head and bid them not flatter themselves and often in the day used to tell them, ' Believe me, my dear children, it won't do ; at twenty-five I might have struggled through it, but at fifty-five I cannot resist.'

"On Thursday the Queen's vomitings returned with as much violence as ever and in the afternoon one of the guts burst in such a manner that all her excrement came out of the wound in her belly, though the surgeons could not by any probing certainly tell whereabouts in the gut the fracture was. The running at the wound was in such immense quantities that it went all through the quilts of the bed and flowed all over the floor.

"Some ignorant people about her who knew not from what cause this evacuation proceeded told the Queen they hoped the relief would do her good, to which the Queen replied very calmly, she hoped so too for that it was all the evacuations she should ever have.

"Every day once, at least, and sometimes oftener from the first of her being under the surgeons' hands they were forced, or thought themselves so, to make some new incision; and before every operation of this kind which she underwent, she always used to ask the King if he approved what the surgeons proposed to do; and when he said they had told him it was necessary and that he hoped she would consent to anything they thought so she always submitted immediately and with the utmost patience, resignation and resolution suffered them to cut and probe as deep and as long as they thought fit. She asked Ranby once, whilst he was dressing her wound, if he would not be glad to be officiating in the same manner to his own old cross wife that he hated so much; and if any involuntary groans or complainings broke from her during the operations, she used immediately after to bid the surgeons not mind her, and would make them apologies for interrupting them with her silly complaints when she knew they were doing all they could to help her.

"From the time of the bursting of the gut the physicians and surgeons who had hitherto, without any disguise or reserve, talked over all the particulars of the Queen's case to anybody that asked them any questions were absolutely forbidden by the King to reveal this circumstance or to give any other answer for the future to anybody whatever who inquired concerning the Queen's health than the general one of her being much as she was.

"During this time the King talked perpetually to Lord Hervey, the physicians and surgeons and his children of the Queen's good qualities, his fondness for her, his anxiety for her welfare and the irreparable loss her death would be to him, yet so unaccountable were the sudden sallies of his temper and so little was he able or willing to command them that in the midst of all this flow of tenderness he hardly ever went into her room that he did not snub her for something or other she said or did. When her constant uneasiness from the sickness in her stomach and the soreness of her wound made her shift her posture every minute, he would say to her; 'How the devil should you sleep when you will never lie still a moment? You want to rest and the doctors tell you nothing can do you so much good and yet you are always moving about. Nobody can sleep in that manner, and that is always your way; you never take the proper method to get what you want and then you wonder you have it not.' Notwithstanding the constant pain she was in and her great want of rest the physicians never gave her opium but one night. She herself was not much inclined to take it and the physicians, thinking that it might possibly, from its binding quality, prevent the relief she so much wanted were not very forward to prescribe it. She had not

rested with it all night and when the King came into her room in the morning as she lay with her eyes fixed at a point in the air, as people often do in those situations when they are neither enough at ease to shut their eyes and sleep or to see the things they seem to look at, the King with a loud quick voice said to her; 'Mon Dieu ! qu'est ce que vous regardez ? Comment peut-on fixer ses yeux comme ça ? Vos yeux ressemblent à ceux d'un veau à qui on vient de couper la gorge !'

" The Queen grew so perceptibly weaker every hour that every one she lived was more than was expected. She asked Dr. Tessier on Sunday the 20th. November in the evening with no seeming impatience under any article of her present circumstances but their duration, how long he thought it was possible for all this to last. To which he answered; 'Je crois que votre Majesté sera bientôt soulagée.' And she calmly replied, 'Tant mieux.' About ten o'clock on Sunday night (Nov. 20th. 1737) the King being in bed and asleep on the floor at the feet of the Queen's bed and the Princess Emily in a couch-bed in a corner of the room, the Queen began to rattle in her throat and Mrs. Purcel giving the alarm that she was expiring, all in the room started up. Princess Caroline was sent for and Lord Hervey but before the last arrived the Queen was just dead. All she said before she died was ; 'I have now got an asthma. Open the window.' Then she said; 'Pray.' Upon which Princess Emily began to read some prayers. of which she scarcely repeated ten words before the Queen expired. The Princess Caroline held a looking-glass to her lips and finding there was not the least damp upon it, cried; ''Tis over'; and said not one word more nor shed as yet one tear on the arrival of a misfortune the dread of which had cost her so many.

" The King went to his own side and as soon as he was in bed sent for Lord Hervey to come and sit by him, where, after talking for some time and more calmly than one could have expected, of the manner of the Queen's death, he dismissed Lord Hervey and sent for one of his pages to sit up in his room all night, which order he repeated for several days afterwards. And, by the bye, as he ordered one of them for some time after the death of the Queen to lie in his room and that I am very sure he believed many stories of ghosts and witches and apparitions, I take this order to have been the result of the same way of thinking that makes many weak minds fancy themselves more secure from supernatural danger in the light than in the dark and in company than alone."

John Ranby (1703–73) who operated upon the Queen. was appointed Surgeon in Ordinary to the King's Household in 1738 and Serjeant Surgeon in 1740. He was a Londoner by birth and was apprenticed to Edward Bernard. Like his master, he was a 'foreign brother' of the United Company of Barbers and Surgeons and never held office. When the United Company was dissolved in 1745 he was elected the first Master of the Surgeon's Company, serving a second time in 1751. In memory of his first Mastership he presented the Surgeons' Company with the loving cup which is now in the possession of the Royal College of Surgeons of England. By the kind permission of the President and Council, it is here represented. It is a standing cup of silver with a cover and measures 15½ in. in height and 7½ in. diameter at the rim. The inscription runs :—

Die Julii primo MDCCXLV
Hoc qualecunque suae in Fratres
Observantiæ Monumentum
Dignissimæ Chirurgorum
Londinensium Societati
Consecrat Joannes Ranby

which may be translated, " John Ranby dedicates this memorial, such as it is, to the very worshipful Company of Surgeons on the first day of July, 1745, as a token of regard for his brethren." The armorial bearings on the side of the cup opposite the inscription appear to be a later addition, for they were not granted until 1822. It is probable, therefore, that they were engraved in that year when the new Charter gave the College a Mace and the right to call itself The Royal College of Surgeons of England. The hall-mark shows

that the cup was made in London, 1745, and the maker's mark, E. G. with a full stop above and below in a lozenge, that it was produced by Elizabeth Godfrey.

Ranby owned land at Fulbrook, Ealing, jointly with Henry Fielding, the novelist, who mentions him in *Tom Jones*. He succeeded William Cheselden as Surgeon to the Royal Hospital at Chelsea. His appointment is dated May 13, 1752. He died there on Aug. 28, 1773, and is buried under a plain altar tomb on the north side of the burial ground. Ranby married in 1729 Jane, elder daughter of the Honourable Dacre Barrett-Leonard, but had no children by her. His natural son, John Osborne (1743–1820), took the name of John Ranby in 1756.

Ranby is described as a man of strong passions, coarse voice, and vulgar manners. His chief work was *The Method of Treating Gunshot Wounds*, a small 12mo which appeared in 1744, a second edition being published in 1750, and a third in 1781. It gives an account of some of the cases he saw when he was serving under Lord Stair in the German campaign which ended with the battle of Dettingen in 1743, where he had the Duke of Cumberland as a patient. It is written in an extremely simple style and foreshadows that associated aid for the wounded which has only lately been adopted.

The two physicians called Tessier and Broxholme by Lord Hervey were Physicians to St. George's Hospital, then newly founded as an offshoot from Westminster Hospital.

George Lewis Teissier was a foreigner who graduated at Leyden in 1710, and, as his estate was administered by his sister living at Zell, he was probably a German by birth. He was appointed Physician to the Household of King George I in 1710, and attended several members of the family of George II. He lived in Pall Mall and died there unmarried on May 22, 1742. Mr. George C. Peachey has shown that he owed much to Sir Hans Sloane.

Noel Broxolme was born at Oakham, Rutlandshire, in 1686, was admitted a King's Scholar at Westminster School in 1700, and matriculated as a Junior Student from Christ Church, Oxford, in 1704. He was elected the first Radcliffe Travelling Fellow in 1715. Whilst he was living in Paris a grateful patient gave him £500 stock in the Mississippi Scheme which he sold shortly afterwards for £10,000. He practised for some years in Oxford, and in 1730 married the wealthy widow of William Dowdeswell, of Pull Court, Worcestershire. He was elected Physician to St. George's Hospital in 1733, and in the following year was appointed Physician to Frederic, Prince of Wales. Horace Walpole, writing to George Montagu under the date July 25, 1748, says : " You have seen in the papers that Dr. Broxholme is dead. He cut his throat. He always was nervous and vapoured and so good-natured that he left off his practice from not being able to bear seeing so many melancholy objects. I remember him with as much wit as ever I knew ; there was a pretty correspondence of Latin odes that passed between him and Hodges." He had no children by his wife, who survived him with his two stepsons. He lived in Albemarle Street but died at Isleworth in July, 1748.

The Portrait of Queen Caroline that appeared in the last issue is copied from that in the National Portrait Gallery, painted by Enoch Seeman (1674–1744).

CALCAREOUS DEPOSITS IN THE SUPRASPINATUS TENDON.

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THE occurrence in X-ray photographs of the shoulder of shadows situated over the top of the great tuberosity was first recognized by Painter,[1] and thought by him to be due to thickening in the subdeltoid bursa. In four cases in which such shadows were present and which he explored by operation he found in two a cheesy material having the appearance of the contents of a wen which he thought was contained in the subdeltoid bursa. But Codman,[2] who was present at one of these operations, stated that this material was not in, but deep to, the bursa. According to him a small opening was made into the bursa sufficient to admit the forefinger; the interior was not abnormal except for some firm adhesions which made it difficult to separate the plane of the bursa from the neighbourhood of the bicipital groove and anterior part of the great tuberosity. With the finger in this opening it was found that during rotation of the humerus a mass corresponding to the X-ray shadow could be felt to pass under the finger. This mass lay on the great tuberosity. An incision into it allowed the escape of about half a drachm of material resembling the contents of a dermoid cyst. Codman stated that he had seen another case in which there was calcareous material in the same situation and that he was familiar with the appearance in X-rays.

The subdeltoid, or subacromial, bursa is mentioned in Quain's *Anatomy* without description. Cunningham describes it as " a bursal sac as large as a hen's egg which is sometimes divided into loculi." From personal observation I should describe it as a tissue space lying in areolar tissue, some strands of which cross the cavity and may divide it into different compartments. It appears, however, to have no definite lining, and it has not shown any sign of being inflamed in any of the cases described below except *Case* 7, in which the deltoid muscle was adherent to the great tuberosity.

'Subdeltoid bursitis' has been very extensively discussed by Codman, who quotes many other varieties apart from the two cases in which calcareous matter was found. He believes that partial rupture of the supraspinatus tendon is a common lesion and that the calcareous formation may result from an insertion fracture of this tendon. Since Codman's description many cases of this calcareous deposit have been recognized and described. Brickner[3] has published a very thorough description based on a series of seven operations.

CASE REPORTS.

The following cases have come personally under my care :—

Case 1.—A woman, age 35, had had a pain in the left shoulder six or seven years previously which had never completely cleared up, so that she was unable to

play golf comfortably. She then wrenched the shoulder badly while playing cricket. It was very painful, was treated by electrical stimulation of the muscles, and improved in muscle power; but the pain continued. When first seen about four months after the onset of acute symptoms the pain was very severe. Very little abduction was permitted, but rotation of the joint was fairly good. There was a tender spot over the region of the great tuberosity. An X-ray showed a characteristic shadow in this situation. Operation was discussed, but as the patient was at the time five months pregnant it was decided to postpone this. However, three weeks later the pain was so severe that the patient demanded operation. An incision was made along the anterior border of the deltoid, the muscle was lifted back, and the region of the great tuberosity exposed. An evident inflammatory swelling was found adherent to the bone and was dissected

FIG. 69.—*Case* 2. X-ray showing the diffuse shadow above the great tuberosity.

away, including part of the attachment of the supraspinatus tendon. The pain was relieved immediately; the wound healed without complication. Physical treatment was started five weeks after operation and recovery of function was fairly rapid.

Case 2.—A woman of 32 had a history of only three weeks' pain in the right shoulder which was ascribed to a strain whilst playing tennis, but the pain was present before this occurred. An X-ray had been taken and the diagnosis of fracture made. Operation was carried out as in the previous case, the inflamed area being completely excised. Function was fairly good in two months, but not complete until four months after operation. (*Fig.* 69.)

Case 3.—A man, age 50, had a fall on the shoulder fifteen years previously, and from that time onwards had some rheumatic pain in the right shoulder. In the autumn of 1929 he fell from his horse, injured his back, and made the shoulder a little

FIG. 70.—*Case* 3. The calcareous matter forms a more solid-looking mass which might be taken for a loose piece of bone.

painful. He spent the next day shooting, and the pain then became very acute so that morphia was required for the next week, at the end of which time an operation was performed. On the top of the great tuberosity was a yellow patch about ¾ in. in diameter with an acutely inflamed area extending around it. This yellow patch was lifted entire from the humerus and removed. Beneath it a small opening into the bone was discovered from which calcareous granules were curetted. The wound healed satisfactorily, physical treatment was started on the eleventh day, and function was fairly good at the end of two months. (*Fig. 70.*)

Case 4.—A woman, age 34, had a fall from her horse twelve years previously, injuring the right shoulder, which had never completely recovered. Seven years after this she had a severe attack of pain which necessitated complete rest, and from that time the shoulder was always painful when the arm was lifted. A fresh acute attack of pain necessitating the administration of morphia occurred a week before the patient was seen. At the operation the condition seen was very similar to that in *Case 3*, and there was a similar cavity in the humerus. Massage was started after operation, and the restoration of function was rapid. (*Fig. 71.*)

FIG. 71.—*Case 4.* The shadow is slight and partially hidden by the humerus. It might easily be missed.

Case 5.—A woman, age 50, strained the right shoulder five years previously in reaching upwards. From that time on there was always a certain amount of pain. Two months before she was seen she had a sudden severe pain in the shoulder which was sufficiently bad to prevent her sleeping. An X-ray photograph, taken at the time of onset of the acute pain, showed a characteristic shadow of large size. Another, taken two months later, showed the patch considerably smaller. At the operation the top of the great tuberosity was not obviously inflamed, the calcareous patch was only found by incising into it. It was then removed entire with a portion of the supraspinatus tendon down to the bone. The calcareous matter was dried up and surrounded by fibrous tissue. There was no cavity in the bone.

Case 6.—A woman strained her right shoulder five years previously. One year later the arm was again injured by being pulled severely. For the last year the shoulder has been chronically painful. Her movements were full and only painful at one point during elevation. There was a tender spot over the great tuberosity. An X-ray showed a characteristic shadow, and the shoulder was explored. The appearance seen was similar to that in *Case 5*. In this case there was considerable pain after operation and functional recovery was very slow, but the after-care was not carried out under my direction.

Case 7.—A woman, age 40, first began to get pain in the right shoulder eighteen months previously as a result of playing the violin. There was no definite injury. The pain gradually became worse so that she was obliged to give up playing. The movements of the shoulder-joint were fairly good, abduction being free up to the right angle, external rotation quite free, but internal rotation less than in the left

shoulder. There was a tender spot over the great tuberosity, and raising the arm in forward flexion produced the most pain. An X-ray showed a large irregular calcified patch over the great tuberosity. It appeared to be situated rather far back. At the operation an incision was made along the front of the deltoid, and on retracting the muscle backwards it was found to be adherent to the great tuberosity over an area about an inch in diameter. When it was retracted this area was found to be rather prominent and slightly yellow in colour. An elliptical incision was made around it down to the bone, and everything peeled off the bone from before backwards. A small cavity containing inspissated calcareous material was opened because it communicated with a tiny cavity in the bone. In the upper part some fibres of the supraspinatus tendon were seen and removed, but the tendon was not completely divided and the shoulder-joint was not opened. The cavity in the bone was about $\frac{1}{4}$ in. in diameter and $\frac{1}{4}$ in. deep. It was curetted with a small gouge. Recovery was rapid. Physical treatment was started at the end of a fortnight, and movements were practically complete two months after the operation.

Case 8.—In the following case the X-ray appearance was not typical and there is some doubt about the diagnosis. A man, age 55, strained his right arm heaving a kit-bag into a dinghy. The joint was strapped for a fortnight and he then had some physical treatment, but the shoulder did not get right. Two months later the joint was examined under an anæsthetic and no adhesions were found. When seen four months after the original injury he had a painful spot on the great tuberosity, a limitation of all movements to about half their ordinary range, and pain on active abduction. X rays showed an irregularity at the top of the great tuberosity with a doubtful shadow above it. Operation was suggested, but, as the diagnosis was not quite certain and the symptoms were not severe, it was decided to try physical treatment first. The shoulder was treated by Dr. Morton Smart by manipulation and by electrical methods. It improved rapidly, so that four months later the patient was able to carry a gun and use the arm freely, although he still had some aching pain. Another X-ray, taken six months after treatment started, still showed some irregularity on the top of the great tuberosity. Since then the arm has greatly improved. The patient now states that he has free movement but that he cannot take liberties with the shoulder. He has tried to play tennis, but cannot do so. He can do half a day's shooting, but a whole day leaves his arm tired. Recovery is therefore good but not quite complete.

These cases occur in adults of all ages. They can be divided into those which are acute and those which are chronic with acute exacerbation. Injury has not occurred in every case and is often not very definite, but it seems clear that trauma lights up the condition in a case of old standing. In the acute cases the pain may be very severe. In *Cases* 1, 3, and 4 it necessitated the use of morphia continuously.

Amongst the clinical signs the occurrence of a tender spot on the top of the great tuberosity appears to me to be very characteristic, although Brickner in his cases found that it might be absent. Movement may be little restricted in the chronic cases, but when the condition is acute there is much muscular spasm and the arm may be held quite rigid. The X-ray appearance is very characteristic. The shadow may be taken for a loose body in the shoulder-joint. This was so in a case described below. But it is usually situated too far out to be in the joint and, as one radiologist pointed out, there may be a change of shape of the shadow in different positions, suggesting that there is a sac containing opaque fluid rather than a solid piece of bone. It is easy to miss the shadow in some positions of the shoulder because it may lie behind a part of the humerus. In some cases a patch of rarefaction in the great tuberosity may suggest that there is a cavity such as was found in *Cases* 3, 4, and 7.

Case 8 recovered without operation, but the X-ray appearance was such as to make it a little doubtful whether the diagnosis was correct. Such recovery, however, occurred also in the case of a medical man who suffered from the condition and in whom the diagnosis of a loose body in the shoulder-joint was made. The shoulder was explored by another surgeon, but of course no loose body was found. This patient now writes :—

> The only cause I can think of for the trouble was that I rushed up a narrow staircase in the dark, fell forward, and threw out my arm in front to save myself. A few days afterwards the pain was very intense and the only relief came from raising my arm above my head or placing the palm on my occiput. I did not go to bed for about a week, resting for most of the time on a couch. I lost about one and a half stone in weight in a fortnight. G. diagnosed 'myositis ossificans of the deltoid'. X rays showed a foreign body in the shoulder-joint. Nothing found on exploration. G. B., who assisted at the operation, said there was myositis ossificans of the supraspinatus tendon at its insertion in the head of the humerus. Pain after operation continued for a fortnight, but the arm gradually got all right. I still get a good deal of pain at times and there is a lot of grating in the joint and some limitation of movement backwards.

The original X-ray showed a large diffuse shadow over the great tuberosity.

Experience seems to show that removal of the calcareous matter gives the quickest relief from pain in the acute cases and the most satisfactory cure in the chronic cases. The best route is that along the anterior border of the deltoid, as this does not interfere with subsequent function of the muscle.

In the acute cases the appearance seen is very characteristic, with a central yellow patch looking like a thin-walled abscess and obviously inflamed tissue all around. In the chronic cases there is little to be seen wrong, and it may only be possible to identify the exact site of the calcareous matter by an incision into it in the line of the supraspinatous tendon (*Case* 5).

The pathological lesion may be dealt with either by excising it completely down to the bone or by opening and curetting. I have preferred the former as the more thorough procedure. It necessitates opening the shoulder-joint in many cases, but this does not seem to interfere with subsequent recovery. The bone beneath the insertion of the tendon should be examined to make sure that it does not contain a small cavity. I have preferred the incision along the anterior border of the deltoid, lifting this muscle backwards. The approach is a little easier if the incision is made farther back and the deltoid split, but if this is done there is a small risk of interfering with the nerve-supply of the anterior part of the deltoid muscle.

In none of the cases have I found any tear in the supraspinatus tendon or any necrotic tissue.

Pathology.—The material removed was examined histologically in five of the cases. In *Case* 1 (*Fig.* 72), which was very acute, the central cavity contained blood. In its margin lay the calcareous matter, which was non-crystalline and was embedded in masses of inflammatory cells amongst which were a few giant cells resembling those seen in the neighbourhood of a foreign body. Around this central cavity lay fibrous tissue in various stages of development containing acutely inflamed patches, in some of which more

calcareous particles were present. On one side of the central cavity beyond this inflammatory area there was a layer of tendon tissue. In *Case* 2 the appearances were almost identical.

In *Case* 3 (*Fig.* 73) the central cavity was surrounded by a layer of fibrous tissue, the inflammatory tissue lying external to this. In *Case* 4 the central cavity was full of calcareous matter and débris. Around it was a very cellular inflammatory tissue and beyond this on the superficial aspect a definite layer of tendon. On the deep aspect some fragments of bone and cartilage

FIG. 72.—*Case* 1. Section of the margin of the cavity showing the calcareous matter darkly stained, some of which lies in the cavity, some in inflammatory and fibrous tissue around.

FIG. 73.—*Case* 3. Section of the margin of the cyst showing tendinous tissue in the edge of the cavity and inflammatory tissue around with calcareous material both in the cavity and in the inflammatory tissue.

represented a point at which the tendon had been lifted from the great tuberosity with the sharp rugine. In *Case* 6, which was of a more chronic nature, there was no very definite central cavity, the calcareous matter lying embedded in fibrous tissue which did not show any of the evidences of acute inflammation seen in the other sections.

Professor Kettle has looked over these sections, and agrees that they show the changes that would be expected in an acute or chronic inflammation around calcareous matter acting as a foreign body. There is nothing in them to suggest necrosis occurring in the tissues.

Cultures taken at the operations were all sterile, and none of the sections suggests a tuberculous infection. The calcareous material obtained from the first case was examined by Dr. McKenzie Wallis, who found it to consist of calcium stearate. Analyses have been reported by Painter, who states that the 'ash' contained a small amount of sulphate of potash and 90 per cent calcium carbonate and calcium phosphate; and by Brickner, who found chiefly calcium oxalate.

As these observations seemed to be at variance with one another the material obtained from *Case* 7 was carefully investigated by Dr. G. A. Harrison, with the following results :—

Chemical Examination of Cyst Contents.—The cyst contents were white and cheesy in appearance with hard gritty lumps in suspension.

Qualitative tests revealed a small proportion of organic matter (presumably protein mainly). The ash consisted largely of calcium phosphate, but there was also a small amount of carbonate.

Quantitative examination gave the following results :—
Matter soluble in ether : nil.
Matter soluble in hot amyl alcohol : nil.
Matter soluble in ether after acid hydrolysis = 7 mgrm. This gave no reaction for cholesterol. The amount was too small for identification. In view of the fact that hot amyl alcohol extracted nothing, calcium soaps were absent.
The dry weight of cyst contents examined (approximately half) = 231·7 mgrm.
Weight of ash = 162 mgrm.
Weight of calcium (as Ca) = 51 mgrm.
Weight of inorganic phosphorus (as P) = 23 mgrm.

Calculated Results.—Assuming that all the phosphorus was present as $Ca_3(PO_4)_2$, and the residual calcium was combined as carbonate, the following are obtained by calculation :—

Calcium phosphate	=	116 mgrm.
Calcium carbonate	=	15 mgrm.
Undetermined ash	=	31 mgrm.
Total ash	=	162 mgrm.

The nature of these calcium deposits is still uncertain. Nothing similar has been described elsewhere in the body, so it would appear that some local anatomical lesion is responsible for their occurrence. The most obvious suggestion is that of a tear of the insertion of the supraspinatus with a resulting effusion of blood and of some fatty tissue from the interior of the cancellous spaces, that calcification occurs in this, and that the calcareous matter then acts as an irritant foreign body.

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[1] PAINTER, *Boston Med. and Surg. Jour.*, 1907, clvi, 345.
[2] CODMAN, *Ibid.*, 1908, clix. Oct. 22 and following numbers ; 1911, clxv, 115.
[3] BRICKNER, *Amer. Jour. Med. Sci.*, 1915, cxlix, 351.

AN ANALYSIS OF THE MORTALITY
IN ACUTE APPENDICITIS WITH RESPECT TO DRAINAGE
AND THE VARIETY OF OPERATION.

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DURING the period January, 1911 to June, 1923 (twelve and a half years),. accurate notes are found of 632 cases of acute appendicitis operated on in Sir John Marnoch's ward in the Aberdeen Royal Infirmary. From June, 1923, to December, 1929 (five and a half years), 781 consecutive cases were operated on from the ward allotted to Mr. Colt. From the total number (1413) five cases in which the details are insufficient have been excluded from
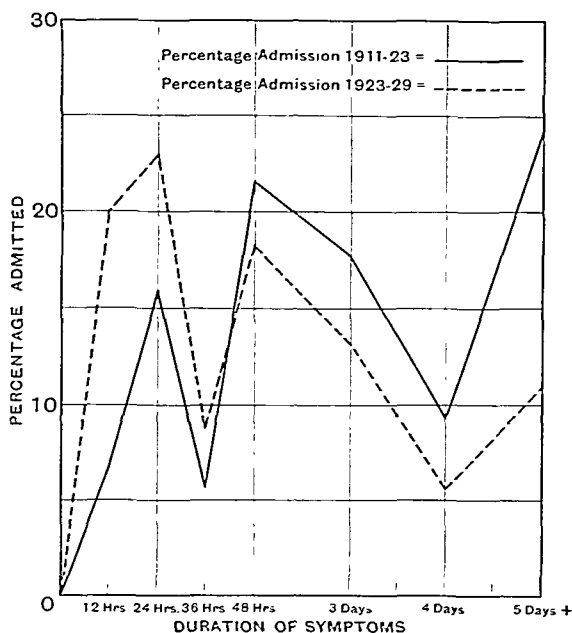
FIG. 74.—Graph showing the percentage admission in 1911–23 as compared with that in 1923–29 with regard to duration of symptoms.

Table I. Two cases of death during anæsthesia have been totally excluded. Owing to the subject of acute appendicitis becoming better known. the 1923–29· cases were on the whole sent in earlier then those for 1911–23, as shown

in the graph (*Fig.* 74), which gives the percentage admission with respect to duration of symptoms.

Table I.—ACUTE APPENDICITIS. NUMBERS OF CASES DRAINED AND NOT DRAINED, 1911–29.

CONDITION OF APPENDIX	DRAINAGE	NO. OF CASES	NO. OF DEATHS	MORTALITY
'Acute with lymph', including 'pus in lumen' and 'gangrene of mucous membrane'	Yes	73	3	Per Cent 4·11
	No	615	9	1·46
Gangrenous not perforated	Yes	119	9	7·56
	No	134	3	2·24
Gangrenous perforated	Yes	105	6	5·71
	No	28	0	0·00
All cases, including abscess and general peritonitis	Yes	609	65	10·67
	No	799	13	1·63

From time to time statistics are published tending to support the advantage of non-drainage of the peritoneal cavity in the various stages of the disease. The figures in *Table I* show the numbers of cases drained and not
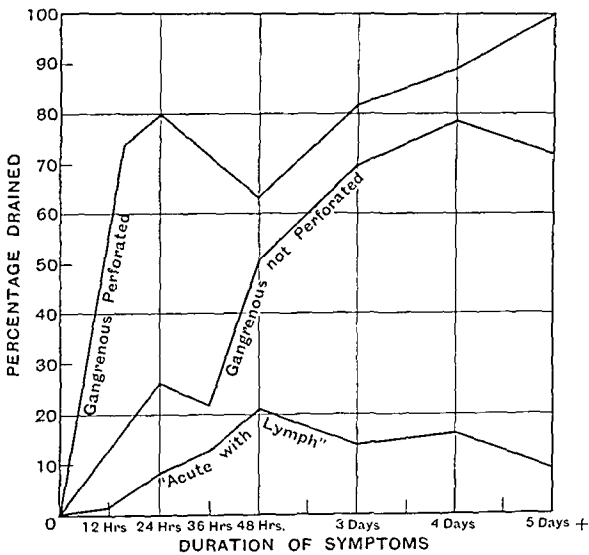
FIG. 75.—Percentages drained and duration of symptoms. (Abscess, general peritonitis, and 'duration not recorded' cases excluded.)

drained, and the corresponding rates of mortality for the period 1911–29. A superficial consideration of the table lends support to those who prefer to

dispense with drainage. A closer inspection, however, shows that such support is not warranted, and it is apparent that any such collection of heterogeneous data is not capable of mathematical analysis. While the material was being arranged it became evident that in general the worse the case the more frequently was drainage employed. This comes out clearly in *Table II* and *Fig. 75*. The table also shows that only a small proportion of 'acute with lymph' cases are drained as compared with the 'gangrenous perforated'.

Tables II.—PERCENTAGE OF CASES DRAINED AND THE MORTALITY IN THE DIFFERENT CONDITIONS OF THE APPENDIX FOUND AT OPERATION, CLASSIFIED ACCORDING TO THE DURATION OF SYMPTOMS.

DURATION OF SYMPTOMS	'ACUTE WITH LYMPH', INCLUDING 'PUS IN LUMEN' AND 'GANGRENE OF MUCOUS MEMBRANE'			GANGRENOUS NOT PERFORATED			GANGRENOUS PERFORATED		
	Drained	Not Drained	Per Cent Drained	Drained	Not Drained	Per Cent Drained	Drained	Not Drained	Per Cent Drained
12 hours	3	156	1·89	2	14	12·50	6	2	75·00
24 hours	15	160	8·57	16	44	26·67	12	3	80·00
36 hours	7	47	12·96	5	18	21·74	10	4	71·43
48 hours	24	92	20·69	32	32	50·00	20	12	62·50
3 days	9	54	14·29	36	16	69·23	22	5	81·48
4 days	7	37	15·91	11	3	78·57	16	2	88·89
5 days+	6	60	9·09	13	5	72·22	18	0	100·00
Totals	71	606	10·69	115	132	46·56	104	28	78·79

Table III.—PERCENTAGE MORTALITY AT DIFFERENT PERIODS OF THE DISEASE.*

	12 Hours	24 Hours	36 Hours	48 Hours	3 Days	4 Days	5 Days +	All Cases
Number of cases	183	250	91	212	142	76	102	1056
Deaths	1	4	2	4	9	3	7	30
Per cent mortality	0·51	1·60	2·20	1·88	6·34	3·95	6·86	2·84

* Abscess, general peritonitis, and ' duration of symptoms not recorded' cases have been excluded.

In the course of classification into more special categories, cases of various degrees of severity were seen to fall into the same class. For instance, it may happen that a patient with fulminating obstructive appendicitis in which the whole appendix has become completely gangrenous in seven hours, as occurred in a recent case, falls into the same group as a relatively mild case of obstructive gangrenous appendicitis with symptoms of the same duration. Again, when peritonitis appears, the extent of it is almost impossible to estimate with any degree of accuracy. Turbid fluid may be sterile, or it may contain organisms of great virulence, and, even when judgement is exercised

in the matter of drainage, chance plays a large part. In fact, only if preference were exercised in cases of apparently the same degree of severity, so that one was drained and the next was not, would the resulting data be of any value. This is a condition which clearly cannot be caried out in practice. It would appear that any opinion or impression that is advanced on the question of drainage is a purely personal one and cannot be supported mathematically until much more exact data are available.

When, however, we come to consider the results in the light of the method of approach to the septic focus, the old surgical rule of employing a short and simple route is found to be justified. *Table IV* shows that, during the years 1911–23, the mortality with the McBurney incision was greater than that with the Battle at practically all stages of the disease. The number of cases operated on through the paracentral incision during this period is unfortunately too small for convenient comparison when the cases are classified according to duration of symptoms. *Table V*, however, gives a fair comparison between the Battle and paracentral incisions, the mortality with the paracentral being greater throughout the series.

Table VI shows the differences in the percentage mortality for the Battle, McBurney, and paracentral incisions, and their statistical interpretation. From these figures it is clear that the Battle incision is preferable. During

*Table IV.—*Acute Appendicitis, 1911–23. Percentage Mortality

Duration of symptoms	12 Hours			24 Hours			36 Hours			48 Hours		
	Number	Died	Per Cent M.	Number	Died	Per Cent M.	Number	Died	Per Cent M.	Number	Died	Per Ce. M.
Battle	21	—	—	44	—	—	23	—	—	50	2	4·00
McBurney	16	—	—	42	3	7·14	6	—	—	54	4	7·41
Paracentral	2	—	—	4	—	—	2	—	—	13	3	23·08

Not included—46 cases with 2 deaths where there was no record of incision;

*Table V.—*Acute Appendicitis, 1923–29. Percentage Mortality

Duration of symptoms	12 Hours			24 Hours			36 Hours			48 Hours		
	Number	Died	Per Cent M.	Number	Died	Per Cent M.	Number	Died	Per Cent M.	Number	Died	Per Cei M.
Battle	20	—	—	30	—	—	18	—	—	36	—	—
Paracentral	133	3	2·26	148	4	2·70	49	2	4·08	105	7	6·67

Not included—10 cases where there

* Fourteen days' history, general peritoni:

the period 1911–23, the chances were 18 to 1 in favour of the Battle as against the paracentral incision, but this mathematically is only 'possibly significant'. From 1923–29, however, the chances, 123 to 1, are found to be definitely in favour of the Battle incision. Again, over the whole period 1911–29 the mortality with Battle's incision was significantly less. It is of interest to note that in the 1923–29 series the diminution in mortality by the paracentral route has not kept pace with that by the Battle method, although practised 4·40 times as often, and about equally by the two surgeons concerned in the second series.

Mathematically considered, an error arises because it cannot be said that the various incisions are altogether randomly employed. Individual preference is exercised, and in course of time practice improves the handling of a case by one particular route. In spite of this, the chances in favour of the Battle incision are so great that this incision should be borne in mind in practice and in teaching by surgeons wedded to the paracentral route.

The low mortality for the Battle incision throughout the whole of the second series is striking. Had not a fourteen-day case with general peritonitis been operated on through this incision, the mortality would have been nil in the 179 cases. With the paracentral incision there is a distinct risk of spreading infection. Visible pus may be packed off, but no one can be sure of packing off

CORDING TO DURATION OF SYMPTOMS AND INCISIONS USED.

3 DAYS			4 DAYS			5 DAYS +			NO RECORD DURATION			TOTALS		
mber	Died	Per Cent M.	Number	Died	Per Cent M	Number	Died	Per Cent M.	Number	Died	Per Cent M.	Total	Died	Per Cent M.
34	2	5·88	26	2	7·69	52	4	7·69	9	1	11·12	259	11	4·25
54	5	9·26	21	—	—	45	6	13·34	12	—	—	250	18	7·20
11	1	9·09	5	1	20·00	26	1	3·85	5	1	20·00	68	7	10·29
8 cases with 1 death where another incision was used.												577	36	6·24

CORDING TO DURATION OF SYMPTOMS AND INCISIONS USED.

3 DAYS			4 DAYS			5 DAYS −			NO RECORD DURATION			TOTALS		
mber	Died	Per Cent M.	Number	Died	Per Cent M.	Number	Died	Per Cent M.	Number	Died	Per Cent M.	Totals	Died	Per Cent M.
21	—	—	4	—	—	15	1*	6·67	—	—	—	144	1	0·69
33	11	13·25	39	4	10·26	69	7	10·14	2	—	—	628	38	6·05
was no record of incision.												772	39	5·05

operation, appendix not removed.

Table VI.—Acute Appendicitis.   Statistical Comparison Between the Incisions Used.*

	DATE	INCISIONS	CASES	DEATHS	PER CENTAGE MORTALITY	DIFFERENCE IN PERCENTAGE (p_1-p_2)	STANDARD ERROR OF DIFFERENCE (E)	$\frac{P_1-P_2}{E}$	CHANCES	MATHEMATICAL SIGNIFICANCE
Battle v. Paracentral	1911–23	Battle	259	11	4·25	6·04	3·11	1·94	18 to 1 in favour of Battle	Possibly significant
		Paracentral	68	7	10·29					
	1923–29†	Battle	144	1	0·69	5·36	2·02	2·65	123 to 1 in favour of Battle	Significant
		Paracentral	628	38	6·05					
	1911–29	Battle	403	12	2·98	3·49	1·39	2·51	82 to 1 in favour of Battle	Significant
		Paracentral	696	45	6·47					
Battle v. McBurney	1911–23	Battle	259	11	4·25	2·95	3·63	1·44	5·7 to 1 in favour of Battle	Not significant
		McBurney	250	18	7·20					
McBurney v. Paracentral	1911–23	McBurney	250	18	7·20	3·09	3·68	0·84	1·5 to 1 in favour of McBurney	Not significant
		Paracentral	68	7	10·29					

* Since *Table VI* was compiled the figures for 1930 and 1931 have been worked out (*Table VII*).   They fully support the previous work.
† In the series 1923–29 the McBurney incision was not used.

Table VII.—Statistical Comparison between Battle and Paracentral
Incisions when the Figures for 1930 and 1931, now
Available, are Added.

	DATE	INCISION USED	CASES	DEATHS	PERCENTAGE MORTALITY	$\frac{P_1-P_2}{E}$	CHANCES	SIGNIFICANCE
Battle v. Paracentral	1923–31	Battle	179	1	0·56	3·0	369 to 1 against paracentral	Distinctly significant
		Paracentral	843	50*	5·93			
	1911–31	Battle	438	12	2·74	2·75	167 to 1 against paracentral	Significant
		Paracentral	911	57*	6·26			

* In two cases suppurative pylephlebitis was present on admission.

invisible bacteria.   It is a matter of chance whether the bacteria or the antibodies are in the ascendant.   If the former, the patient gets worse; if the latter, he improves.   It is tempting to argue that the employment of drainage will do good in the former and will do no harm in the latter, so that the majority of surgeons use a drain in case of doubt.   The risk of a drainage tube causing actual infection must be very small, judging by its use in other. abdominal

cases such as in cholecystectomies. A joint factor that weighs is the difficulty sometimes experienced in diagnosing appendicitis in the early acute abdomen, especially in women, so that the paracentral incision is more often used. A distinct additional risk would appear to be taken in this way when acute appendicitis is actually found. This points to the need for a close study of the early case. On the other hand, when the signs indicate a median pelvic position for the appendix, the short, subumbilical, paracentral incision should prove safer than Battle's.

SUMMARY.

1. Statistical analysis of 1408 cases of acute appendicitis fails to justify any conclusion for or against drainage of the peritoneal cavity. This is a negative conclusion and indicates the difficulty of the subject.

2. With regard to the incisions employed, we find that much the lowest mortality occurs with the Battle incision. This should be the method of choice, except when there is doubt as to the nature of the case, notably in women, or when the appendix is median and pelvic in position.

regarded as a mild obstruction of the upper part of the small intestine, or perhaps a gall-bladder lesion. Subsequently these attacks recurred ; and finally when I operated on him some months later the following condition was found :—

The omentum was quite devoid of any fat and had become thin and of white veil-like structure studded with tiny tubercles ; the intestines were covered here and there with opaque, rigid, white plaques ; the lacteals were very prominent as if they were dilated ; the intestine was slightly contracted in the region of the plaques ; the gall-bladder and stomach were spotted with patches of this membrane.
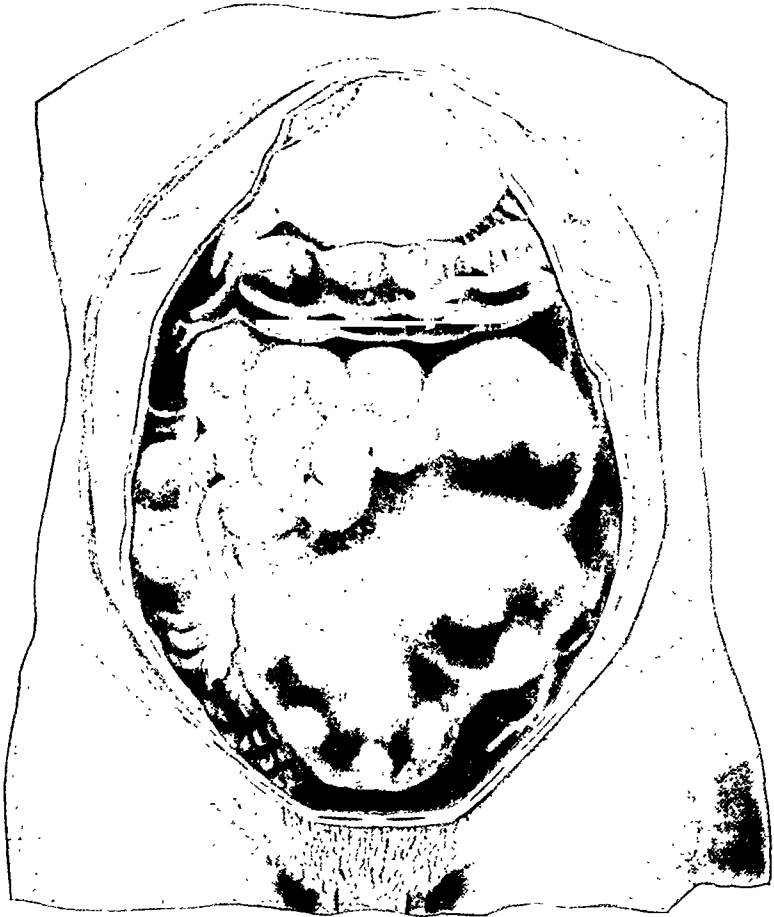
FIG. 76.—Tumour-like masses causing intestinal obstruction in a case of encapsulating chronic peritonitis. Stomach and small intestines are covered with a thick opaque contracting membrane. The colon is normal.

The rest of the organs were normal. There was no definite obstruction, and the attacks of pain were probably caused by painful intestinal peristalsis, resulting from the presence of the rigid plaques.

Pieces of the opaque membrane which were removed proved on microscopic examination to be a hyaloid membrane, having no definite structure and looking like degenerated fibrin. Section of the omentum showed degenerate structureless areas, corresponding to the tubercles. Taking the microscopic and macroscopic characters in conjunction, the condition looked like a patchy tubercular peritonitis in an early stage.

After this operation the patient was comparatively well for nine months, and then he began to get attacks of cramping abdominal pains, which were always associated with constipation. These gradually became more frequent and more severe, till one very bad attack which developed into an acute abdominal crisis. His pains became very severe ; he continuously vomited large quantities of brownish-yellow fluid ; and repeated enemas elicited neither flatus nor fæces. A tender, ovoid tumour the size of a child's head could be palpated in the left upper quadrant of the abdomen. It was obvious that the patient was suffering from an acute obstruction of the upper part of the small intestine, and that an operation was urgently necessary.
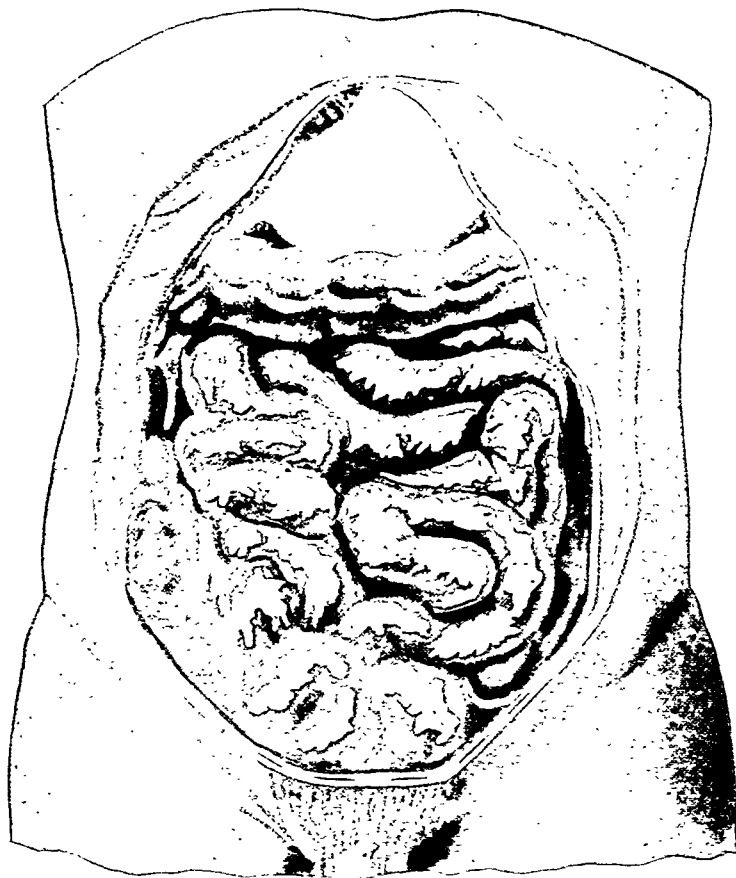
Fig. 77.—The intestines are shown after decapsulation ; the white opaque patches on the antiperistaltic section of the gut cannot be stripped off ; the dark parts are raw, bleeding serosa.

The pathological conditions found at the operations were as follows : The parietal peritoneum was about 2 mm. thick, and it was found difficult to open into the peritoneal cavity because of the subjacent adherent loops of small bowel and the extensive adhesion of these to one another. The extent of the peritoneal cavity was limited by these adhesions, and it contained a small quantity of straw-coloured fluid. The visceral peritoneum of the liver was uniformly adherent to the parietal peritoneum. The colon was normal. The abdominal glands were not affected.

The stomach and the small intestine were almost uniformly covered with an opaque, milk-white membrane 2 to 3 mm. thick, which here and there became thickened into cartilaginous plaques. The membrane had encapsulated groups of small intestinal loops into three smooth ovoid tumours, as illustrated in *Fig.* 76. The upper one, made up of jejunum, was the site of the obstruction, for the segment of jejunum entering it was hugely dilated. I was thus faced with the very great problem of determining how to deal with this high obstruction. My experience showed me that entero-anastomosis could not be done with safety. Even if this were practical, it was possible that obstruction was also present in the other incarcerated intestinal loops, because the jejunal obstruction would prevent it from becoming obvious ; furthermore, it was to be expected that the contractile membrane which had already formed the tumour was very likely to produce obstruction also. It was therefore obvious that the intestine had to be decapsulated—apparently a very formidable proceeding. After some prospecting it was found that the membrane was firmly adherent to the mesentery and to the antimesenteric border of the bowel, and comparatively loosely adherent where the peritoneum is reflected on to the bowel. At this spot I found a line of cleavage when the membrane was incised, which I followed, and by a process of peeling, dissecting, and unravelling the intestines were liberated. When this was done the whole of the intestine presented the appearance shown in *Fig.* 77. The raised white plaques are the places where the membrane could not be separated from the intestine. The dark parts are red, bleeding surfaces of intestine, which is altered serosa because this false membrane is superimposed on the serous membrane.

The wound in the abdominal wall was closed, and the patient made an uneventful recovery.

As the genesis of this peculiar pathological condition was uncertain, it was decided to exclude syphilis as a cause. Accordingly a Wassermann test by four different methods was made, and it proved negative.

Dr. Andrew Brenan, who made the microscopic examination of the membrane reported that the section showed " young fibroplastic cells with fairly plump nuclei."

About three months later the patient, who had been comparatively well, stated that a lump was developing in his abdomen and that he was again becoming constipated. Examination revealed that another of these encapsuled aggregations of intestines was forming. It was now decided to try the effect of deep X-ray therapy, and, with this point of view, Dr. Thwaites was consulted. The following were the specifications of the irradiation given to the patient :—

Volts	220,000
Milliamperes	4
Filters	0·5 mm. Zn plus 3 mm. Al plus 3 mm. asbestos
Focal distance	— 40 cm.
Port	Approximately 10 cm. × 8 cm.
Percentage of erythema dose	Approximately 75 per cent.

Special Details.—Tube output continuous and at constant potential. Rays employed homogeneous and of wave-length = 0·014 Å.U. (approximate).

Gradually, over a period of about three months, the tumour in the patient's abdomen disappeared and his bowels attained their normal function.

DISCUSSION.

Occurrence.—There is very little reference in English surgical literature to this rare form of chronic peritonitis. The form of chronic peritonitis described by Fagge and Hale-White alluded to in the beginning of this paper is probably a phase of this affection in which ascites is the predominant symptom. Under the name 'Zuckergussdarm' (icing-sugar intestine), German writers describe a perivisceral chronic hyperplastic peritonitis, which they term a perivisceritis,[1, 3, 5, 7] and which is obviously a similar condition.

Wilmoth and Patel have recently published a most illuminating article on this subject.[13]   To the type of tumour formation which occurred from the envelopment of groups of intestines by this hyperplastic perivisceritis they give the name 'la péritonite chronique encap-
sulante'.   *Fig.* 78, taken from this article, shows the abdomen of one of their patients opened with a median subumbilical incision. One of the tumefactions which this form of chronic peritonitis produces is visible.   This tumour-like mass consisted of a large part of the small intestine, the uterus, and its adnexæ, enveloped by a tough, whitish, firmly-adherent membrane.   A part of the small intestine and the colon was free.   This patient suffered from peri-umbilical and lumbar pain over a period of about five or six years.   At first the pains came on at long intervals, but latterly they had become more frequent and very painful.   A gradually progressive con-stipation had developed into almost complete obstruction, which had necessitated an opera-tion.   The capsule was incised ; a line of cleavage was discovered ; and the membrane was laboriously removed little by little, until

FIG. 78.—Isolated tumour en-closing uterus and part of small intestine. This simulated a pelvic tumour. (*From Wilmoth and Patel.*)

finally it was possible to release the intestine.   In this particular case, the membrane was examined by Professor Lecene, who reported as follows : " Fragments examined have a structure characteristic of a confluent tuber-culosis, evolving in several parts as far as caseation ; typical follicles, epithelial cells, giant cells of Langhans."   *Fig.* 79 is a reproduction of the microscopic section.

FIG. 79.—Microscopic structure of ' la péritonite chronique encapsulante '. (*From Wilmoth and Patel.*)

Esau,[3] who operated on a man 49 years of age with a diagnosis of multiple stenosis of the small intestine, discovered when he made the laparotomy three enormous white, vertical, sausage-shaped tumours coupled together, and contain-ing nearly the whole of the small intes-tine.   *Fig.* 80 shows the macroscopic appearance of this curious lesion.

We were fortunate that in *Case* 1 our observations extended over eleven years, and that in *Case* 2 we were able to study this disease from its very beginning over a period of years.   We are able, therefore, to fill in certain gaps in the pathology of this subject which are missing in its literature.

Undoubtedly the chronic peritonitis described by Fagge and Hale-White

and the 'Zuckerguss' spleen of the German writers are etiologically the same as these chronic peritonitic affections here described by us, the clinical manifestations of which are mainly multiple intestinal stenoses. We have

a parallel relation in the phases of tubercular peritonitis where its principal clinical manifestation may be in one instance ascites, and in another the formation of hyperplastic tubercular tissue with multiple stenoses of the intestine. It is also probable that the 'Zuckergussleber' (icing-sugar liver), and the 'Zuckergussmilz' (icing-sugar spleen) described by the German writers are also only phases of this disease. It is not possible to venture the opinion that the condition called 'polyserositis' in which all the serous membranes, including the pericardium and the pleura, are affected is of the same origin.

Clinical Manifestations and Macroscopic Characters.—It should serve a useful purpose if, from our own observations and with the literature at our disposal, we summarize the main clinical manifestations and macroscopic

FIG. 80.—The appearance of the tumours in Esau's case.

characters of the various phases of this disease in so far as they are concerned with its surgery.

Early Stage.—In this stage the patient may suffer from attacks of colicky peri-umbilical abdominal pain. At first these attacks occur infrequently and at irregular intervals and are associated with constipation. They may simulate attacks of subacute appendicitis. The patient may suffer from a dyspepsia, not unlike the dyspepsia which occurs in certain early infiltrating forms of gastric carcinoma. The patient also suffers from mild, but definite, constitutional symptoms : loss of energy, loss of appetite, tendency to faint, attacks of sweating. The macroscopic characters at this stage were well exemplified by the conditions found at the first operation in *Case* 2. They may be described as follows :—

1. There is an irregular distribution of white, membranous, cartilaginous plaques over the small intestine.

2. In some parts of the intestine these plaques are very thick and are beginning to contract : they do not cause narrowing of the lumen of the bowel. They must, however, interfere with the peristaltic movement and cause it to become painful. They are the pathological basis for the attacks of colicky pain.

3. There is also a patchy, perivisceral distribution of this tough membrane over the stomach. This limits the distensibility of the stomach, probably causing a disorder of the normal painless peristalsis, as well as the dyspepsia.

4. The lesion is strikingly limited to stomach, small intestine, and liver.

At this stage, as indeed at any stage, it is quite impossible to make a diagnosis on the basis of the dyspepsia and the vague abdominal pains. The

question of the presence of an early gastric carcinoma will come into the consideration ; while, on the other hand, the peri-umbilical colicky pain will suggest a diagnosis of appendicitis. A careful X-ray examination in our *Case* 2 gave us no information whatever ; there was no delay in the small intestine.

Middle Stage.—Multiple intestinal stenoses ; multiple tumour formation. In this phase of the disease all the clinical manifestations of an intestinal obstruction of the highest part of the jejunum occur. At this stage tumour formation also takes place. This is likely to be mistaken for a true tumour. The colon may be quite free from the membrane. The conditions disclosed at the second operation on *Case* 2 (*see Fig.* 76) exemplify this stage well. The macroscopic characters are as follows :—

A whitish, tough, opaque, membranous shell, varying from 2 mm. to 5 mm. thick, enveloping uniformly the whole of the small intestine and its mesentery. It is superimposed on the parietal peritoneum and is intimately and inseparably attached to it. This membrane is firmly attached to the deep part of the mesentery and to the intestines, but it is loosely attached where the peritoneum is reflected back from the mesentery on to the intestine. At this part it can be separated from the underlying serosa for a centimetre or two on to the intestine, and a line of cleavage obtained.

Late Stage.—Usually at this stage in addition to the tumour formations the peritoneal cavity is almost entirely obliterated. This is caused by the adhesions of the intestinal loops to one another and to the parietal peritoneum. The colon, as well as the other organs in the abdominal cavity, is now also covered with the membrane, which is now much thicker and more contractile, and so closely united to the serosa that it is separated only with the greatest difficulty. If an incision is made into the intestines at this stage, in the presence of this thick and very contractile membrane, the intestine will practically turn inside out. For this reason, therefore, it is dangerous to perform any anastomosis.

Stage of Isolated Tumours.—Not infrequently the uterus and part of the small intestines may be enclosed with this thick membrane in such a way as to form a tumour just like an ovarian cyst. Wilmoth and Patel[13] describe a case of this sort. *Fig. 78*, taken from their paper " La Péritonite chronique encapsulante ", is an illustration of the condition. The tumour contains the uterus and part of the small intestine, and for a time was mistaken for a fibroma of the uterus.

It is particularly necessary to describe this isolated tumour manifestation, because a surgeon finding such a tumour at operation and not knowing the pathology of it would have great difficulty in dealing with it in the proper manner.

Etiology.—Different authors ascribe different causes for this condition. Fagge and Hale-White, finding it occurring with interstitial nephritis, regard it as a complication of this disease. Other authors regard syphilitic infection as responsible for the cause of this unusual form of chronic peritonitis. In this respect it is significant that the Wassermann test carried out by four different methods was negative in our *Case* 1.

Most French writers look upon it as an obscure, unusual manifestation of

THE REPAIR OF CLEFT PALATE.

By ALEX. MITCHELL, ABERDEEN.

WITH NOTES ON THE ADMINISTRATION OF ANÆSTHETICS.

By J. ROSS MacKENZIE, ABERDEEN.

OF all congenital defects, one of the most serious is a badly cleft palate, and the lot of the unfortunate victim of this deformity is in too many instances made harder when he is subjected to several unsuccessful operations. I cannot think of any operation which, when completed, seems so satisfactory but is so frequently doomed to complete failure. In many instances the failure is an operative one. The suture line which in a few days gives fair promise of success has by the end of a week broken down completely. In other cases in which operation has been done when the child has reached the age of three or four years and the suture has been satisfactory the functional result is poor, for although we have suceeded in uniting the cleft, the soft palate is immobile and drawn forward so that the space between its posterior margin and the pharyngeal wall can never be closed as is necessary for proper speech. What we must aim at is to ensure healing of the cleft with reasonable certainty and with safety in young children before they have begun to speak.

It is an axiom of surgery that the number of methods which have been suggested and practised for the cure of any particular condition is in inverse proportion to the success of any one of them.

For primary operations we have the old Langenbeck operation, the flap operation of Lane, and the Brophy method of forcible approximation of the cleft. In this country the Langenbeck operation as practised and popularized by Sir James Berry and others, has given good results in the hands of some, but it must be admitted that the general results are not good. The utilization by Veau[1] of the mucous membrane of the septum, Wardill's[2] plan of bringing forward the posterior wall of the pharynx to meet the shortened soft palate, and the Gillies-Fry[3] operation which aims at a perfect anatomical and functional result as regards the soft palate—leaving the cleft in the hard palate to the care of the dentist—are all methods which, although they have been successful in the skilled hands of their originators, are not likely to be, and in fact have not been, so successful in the hands of the general surgeon.

Operations which demand special skill in plastic work, considerable expenditure of time, and some risk to the lives of young and fragile children, can never prove a satisfactory solution to this problem.

My own experience extending over about twenty years has resulted in profound dissatisfaction. Unsuccessful attempts at the Lane operation led

to such extensive necrosis of tissue as to make any secondary operation impossible. The Langenbeck operation—the one usually employed—I have been able to perform in what seemed at the time a most satisfactory way— that is, the palate was sutured without injury to flaps and without undue tension ; but in far too big a proportion of cases the suture line gave way badly. During the past year I have adopted a method which I had previously tried without success. It is a two-stage operation (*Figs.* 81–84).
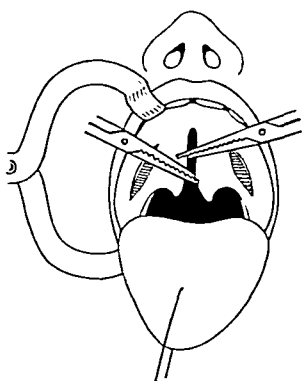
FIG. 81.—End of first stage.
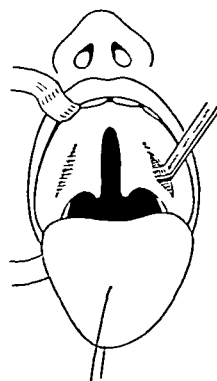
FIG. 82.—Beginning of second stage.
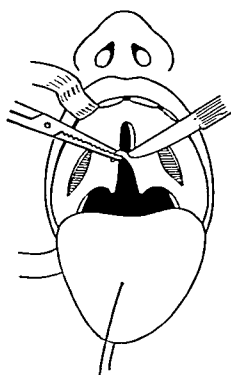
FIG. 83.—Second stage.
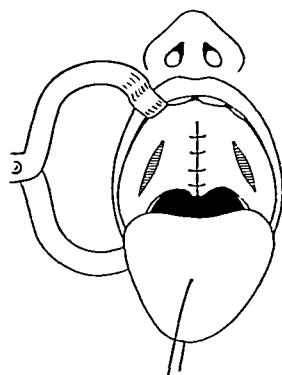
FIG. 84.—End of second stage.

FIGS. 81–84.—OPERATION FOR REPAIR OF CLEFT PALATE.

This method is not a new one and was recently mentioned in a discussion at the Royal Society of Medicine. The first stage is that of separation of the flaps, the second rawing of the edges and suture. Why this operation should not have been successful when tried out some years ago is now difficult to say, but possibly the second stage was done far too soon after the first. Since performing the operation in two stages with an interval of at least two weeks practically complete healing of the suture line has been obtained in all but one case. In order that this method may be carried out with success certain essential points must be observed :—

1. Neither operation should be unduly prolonged, for continuous and unnecessary handling devitalizes the tissues and prevents healing.

2. At no stage of either operation should excessive bleeding be permitted to occur.

3. The flaps should present a healthy and well-vitalized appearance, and there should be no appearance of sepsis in the mouth before the second stage is undertaken.

4. Rectal anæsthesia is the anæsthetic of choice for the second operation and no intratracheal or intranasal anæsthetic tubes should be employed.

First Stage.—For the first stage ordinary anæsthetic, preferably open ether with a preliminary dosage of atropine, is sufficient. The patient's head is dropped over the end of the table, the mouth held open by a simple gag, and the tongue drawn forward by a traction suture placed in the middle line. A small relaxation incision is made external to the posterior palatine artery on either side (*Fig.* 81). A gauze mop is placed on either incision and firm pressure made with the thumbs for two or three minutes to check bleeding. A little more anæsthetic may now be given if necessary. Then with fine elevators the separation of the palate is done, pressure with gauze being again applied. If any blood gets past into the pharynx it is at once removed by a suction tube. The last stage consists of cutting the attachment of the soft palate to the posterior margin of the bony palate. Pressure is re-applied until bleeding has stopped, and finally all the raw surfaces are painted with Whitehead's varnish. This concludes the first stage, which can be performed in about ten minutes.

Second Stage.—The second operation should not be undertaken until at least two weeks later. If there is still an appearance of sepsis in the mouth or if the child's general condition does not seem satisfactory, the operation may be safely postponed for another week. The longest period between the two operations in my cases was three weeks. For the second stage we prefer rectal anæsthesia, which may be supplemented if necessary by nitrous oxide gas and oxygen given through a small intra-oral tube. On examination of the mouth it will be seen that the only trace of the first operation is a narrow scar on either side representing the relaxation incisions (*Fig.* 82). Into each of these scars a palate elevator is gently inserted and it will be found that with very little effort and without fresh bleeding the flaps can be completely separated from the hard palate and brought to meet in the middle line. In some cases a little extension of the original incisions may be advisable. The opposing edges of the flaps are now rawed from end to end and sutures applied (*Figs.* 83, 84). Sutures of fine pliable silkworm gut are inserted by means of small cutting needles and Lane's needle holder. The use of the old-fashioned needles is to be deprecated as they are too broad and cause unnecessary injury to the tissues. The suture line is commenced in front and carried backwards, separate sutures being used in preference to a continuous one. After careful adjustment of the edges a few superficial sutures of six 0 catgut may be inserted. When the last stitch has been placed in the uvula it is drawn forwards so that the posterior surface of the soft palate is exposed and several fine catgut sutures are applied.

During this operation there is very little bleeding, and after it has been stopped by gentle pressure the suture line and the relaxation areas are painted over several times with Whitehead's varnish. Throughout both operations, and especially in the second stage, the greatest care and delicacy must be observed in handling the tissues.

After-treatment. — The following method of after-treatment has been found to be satisfactory :—

After the First Stage.—Rectal salines for three or four days every four hours. Sterile water by mouth for the first night. Sterile fluids, glucose and water, beef tea and chicken tea. No milk for three or four days. then light diet. Younger children have their mouths cleaned with saline solution and glycerin and borax applied later. Children who have been taught before operation to wash out their mouths use a mouth-wash of carbolic acid, liq. potassæ, glycerin, and water.

After the Second Stage.—Rectal salines every four hours for three or four days. Sterile water by mouth for the first day. For the next few days sterile glucose solution, beef tea, and chicken tea. Later on strained soup, fruit drinks, cocoa, and egg drinks until the stitches are removed, usually from the tenth to the fourteenth day. The care of the mouth is as in the first stage—the gums and teeth are swabbed after each feed, but the palate and suture line is not touched, the fluid being allowed to run into the mouth. When a sedative is required nepenthe in appropriate doses has been found most satisfactory.

Results of Operation.—The results of the cases treated by this method are infinitely superior to those I have been able to obtain by completing the operation in one stage, and I feel that what was unboubtedly the most unsatisfactory and disappointing section of my work can now be approached with confidence and with a reasonably sure hope of success. Whatever method of treatment be adopted, there are so many adverse factors at work that failures are bound to occur now and again ; but I am convinced that if the procedure which I have attempted to describe is followed, a surprising percentage of good results will be got. By good results I mean good operative results. The question of ultimate results as to function, i.e., speech, is more difficult, but I think the indication is fairly clear that if we are to have any hope of good functional results we must obtain a good operative result before the child attempts to speak, and that about one year or under is the best age at which to repair the cleft.

My intention is to apply the operative plan which I have outlined to children of about a year old, and by gradually reducing the age at which it is performed to gain more definite information. At present I do not see any reason why good results should not be obtained in children considerably under one year, and I feel that if they could be operated on at say six to nine months the functional results ultimately produced would be very much better than those we see at present.

During the past year I have operated on ten cases, the eldest aged $8\frac{1}{2}$ years and the youngest 11 months. The average age was 2 years and 9 months. In one case in which the cleft was confined to the soft palate only

the suture line broke down completely. In all the others the suture was completely successful except in two cases where an additional stitch had to be subsequently inserted into the tip of the uvula.

I wish to express my thanks to Dr. F. J. T. Bowie for the accompanying sketches, and to Miss Adams, my ward sister, for the great care which she has always displayed in the after-treatment of these cases.

NOTES ON THE ANÆSTHETIC.

In few operations is the surgeon more dependent on the method and the character of the anæsthesia for a clear operation field than in cases of cleft palate. The induction of anæsthesia with ether or with ethyl chloride and ether presents no difficulty and usually suffices for the first stage of the operation, because when necessary the mask may be conveniently re-applied while hæmorrhage is being controlled by pressure in the relaxation incisions.

The problem that confronts the anæsthetist is the maintenance of the anæsthesia during the second stage of the operation. Frequently this is carried out by means of chloroform or a mixture of chloroform and ether, on a towel or in a Junker inhaler, or by endotracheal gas, oxygen, and ether through the nose. These methods are either unsatisfactory to the patient or inconvenient to the surgeon. Frequently at an important stage in the surgical procedure the anæsthetist urgently requires the operation field in order to maintain or to re-induce chloroform anæsthesia. Apart from the immediate risk on the table, chloroform devitalizes the tissues in the operation area and reduces the general recuperative powers of the patient. It is doubtful whether, with rare exceptions, the administration of chloroform to young children is ever justified. Nasal endotracheal catheters passing as they do in close proximity to the operation field, are a disturbing visual element to many surgeons.

In our experience the method of anæsthesia most suitable for the second stage of the operation is colonic oil—ether with paraldehyde. The procedure is as follows : A mild aperient on the morning prior to the day of operation is followed late in the afternoon by an enema and a 5-gr. chloretone suppository. The suppository is repeated on the morning of the operation. Atropine sulphate $\frac{1}{150}$ gr. hypodermically and nembutal $\frac{1}{3}$ gr. to 1 gr. by the mouth is given forty-five minutes before the time of operation. Nembutal appears to be a suitable sedative for infants, and children and induces sleep before and after operation. The mixture for injection consists of equal parts of olive oil and ether, and the dose is reckoned as 1 drachm for every 2 lb. of body weight. One drachm of paraldehyde is added to the dose.

The administration begins thirty minutes before the time of operation and is carried out, preferably with the patient on the operation table in the moderate Trendelenburg position. An elongated funnel is used and the catheter inserted through the anus to a distance just beyond the transverse rectal folds. The administration continues and is supervised by the anæsthetist until no oil-ether mixture returns to the funnel when it is lowered. This usually requires from twenty to thirty minutes.

For a clear operation field and an uninterrupted operation the pharyngeal reflex must be absent, and this necessitates a moderately deep surgical anæsthesia. Any delay in the development of the required depth of anæsthesia for beginning the operation can be corrected by the administration of a few drops of ether on a mask. The absorption of ether from the bowel will then be found to balance the elimination of ether through the pulmonary system and maintain anæsthesia. Any risk to the patient from overdosage or from abnormally rapid absorption of ether with respiratory depression and anoxæmia can be completely controlled by the administration of a mixture of oxygen with 5 or 7 per cent carbon dioxide through a flat, hook-shaped tube hung over the angle of the mouth. The tube should be fixed in position with a strip of adhesive plaster. Further, if it proves necessary, the colonic ether anæsthesia can be supplemented by means of gas, oxygen. and ether through this mouth tube without disturbing the surgeon. On return to the ward, two catheters are passed into the rectum, one of which is attached to a Higginson syringe or to a funnel, and the bowel is washed out with soap and water followed by saline until the odour of ether disappears from the returned fluid.

In conclusion two points require to be emphasized : (1) A history of recent enteritis contra-indicates this form of anæsthesia ; (2) Our experience with colonic ether anæsthesia has been marked by the absence of any post-anæsthetic complications.

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MEGACOLON:
EVIDENCE IN FAVOUR OF A NEUROGENIC ORIGIN.*

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DIVISION of the sympathetic nerves of the colon is becoming increasingly popular as the surgical treatment of choice in cases of megacolon. The exponents of this form of treatment claim merely that the interruption of the sympathetic paths will remove all opposition from such detrusor ability as the dilated bowel enjoys : they do not postulate sympathetic overaction as the cause of the condition. Nevertheless, the remarkable efficacy of lumbar ganglionectomy,[1, 2] and of the simpler and more direct presacral neurectomy of Learmonth and Rankin,[3] demands that an effort be made to produce an experimental variety of megacolon by alteration in the extrinsic nerves of the bowel.

The alternative procedures that at once suggest themselves for this experimental purpose are prolonged stimulation of the sympathetic nerves of the colon, and the division of their parasympathetic antagonists. The latter method, obviously the more practicable, has been chosen here, and the effect on the colon of deprivation of its parasympathetic (sacral) nerves has been observed in a series of cats. The results of this experimental work are presented in this paper, and are correlated with the theories which have been previously formulated. To these is added an anatomical study of the nerves of the colon. The present interest in these colonic nerves renders unnecessary any apology for a repetition of anatomical detail. The pathology of megacolon as it occurs in man has been so closely observed and so fully described that it would be superfluous to discuss it here : attention will be drawn only to certain clinical and pathological phenomena which throw light on the origin of the condition. The figures given are based on 100 fully detailed cases taken at random from the extensive literature which envelopes megacolon, access to which can be gained by reference to the bibliographies compiled by Finney,[4] Barrington-Ward,[5] and Bartle[6] in their comprehensive essays in this subject.

ETIOLOGY OF MEGACOLON.

Megacolon can be defined as dilatation and hypertrophy of a part of the colon or of the whole colon, occurring in the absence of a gross obstructive lesion. It has been customary to divide cases of megacolon into the congenital and acquired varieties, the former presenting symptoms in infancy or

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childhood (in one case appearing so early as the seventh fœtal month), the latter not manifesting itself until adult life, and, most commonly, middle age. The 'acquired' cases may or may not be cases of congenital megacolon in which the condition is of slight enough degree to support life without symptoms during childhood and youth, but in which the dilated, hypertrophied, and overworked colon is the first tissue to succumb to the degeneration of advancing age. In megacolon the pelvic colon is affected alone in 37 per cent of cases, and in 34 per cent the upper part of the rectum or the whole of the rectum also suffers. The dilatation extends proximally to the ileo-cæcal valve in 25 per cent of cases, and in the remainder the descending, transverse, and ascending parts of the colon are involved with decreasing frequency. In 5 per cent of cases an associated dilatation of the bladder is described. Of 4 cases treated recently by Mr. N. M. Dott[7] in the Royal Hospital for Sick Children, Edinburgh, no fewer than 3 showed this complication, with in addition a high residual urine. Since in cases of megacolon the measurement of the residual urine is not a routine part of the investigation, the figure given above for bladder involvement probably underrates its true frequency. It is of interest that the bladder in these 3 cases shared with the colon the improvement which followed presacral sympathectomy.

Theories of Megacolon.—The theories advanced to explain megacolon fall into three groups : (1) Developmental ; (2) Mechanical ; and (3) Neurogenic.

1. *Developmental.*—It has been argued that the condition is a developmental dilatation with secondary hypertrophy (Mya[8]), developmental hypertrophy with secondary dilatation (Fenwick[9] and Genersich[10]), or combined dilatation and hypertrophy of developmental origin (Hirschsprung[11]), but the nature of the developmental error has not been explained.

2. *Mechanical.*—The valves (Wilkie[12]), torsion of a long mesocolon (Bartle[6]), kinking at the pelvirectal junction of a long pelvic colon (Marfan[13]), rectal and anal atresia (Treves[14]), and connective-tissue replacement of the muscle coat of the colon (Concetti[15]), which have been described as responsible for megacolon, are quite as likely to be sequelæ of that condition, and against all these suggestions is the small but definite percentage of cases which present a bladder also dilated. No mechanical change in the colon can be held responsible for a dilated bladder.

3. *Neurogenic.*—Megacolon has been ascribed to reflex spasm of the internal sphincter (Fenwick[9]), non-relaxation of the sphincter (Hurst[16]), paralysis of a bowel segment (Pennato[17]), and it will be observed that all these suggested causal conditions are compatible with a relative sympathetic overaction. Hawkins[18] considered the condition due to neuromuscular inco-ordination, i.e., non-relaxation or contraction of a ring segment at the moment when a peristaltic wave reaches it and demands dilatation. But this is advanced only as an attractive theory and has no evidential support. Monro Cameron[19] has described absence of the cells of Auerbach's plexus at the lower extremity in a case of megacolon, but other observers deny any reduction in the cells of Auerbach's plexus in these cases, and the reduction in the number of cells observed by Monro Cameron may easily be secondary to that chronic fibrosis which has been observed both in the bowel wall and in the mesocolon, the result of stasis in the bowel.

An argument in favour of a neurogenic origin for megacolon is of course the success of sympathectomy in the disease, although, as has been mentioned, the advocates of the operation rightly claim that the operation remains rational even though the nerves they divide are not responsible for the disease. An indisputable argument, however, in favour of a neurogenic origin for the disease is the occasional association with it of a dilated bladder. The only feature which bladder and lower colon share in common is their nerve-supply.

Two of Dott's[7] cases of sympathectomy for megacolon may be cited here. In the first of these the megacolon was associated with a mass of tuberculous glands overlying the presacral nerve, but not directly related to the enlarged bowel. This suggested that the colonic enlargement was due to stimulation of the sympathetic colon-dilator nerves at this point—an example in nature of that prolonged stimulation of a nerve which is so difficult to effect in the laboratory. In the second case the colonic and presacral nerves removed at operation appeared much thicker to the naked eye than those of children of the same age without megacolon. Sections of these nerves, however, when compared with apparently normal controls, showed not a numerical increase in fibres but an increase in epineural tissue. This can be explained as merely part of that connective-tissue excess throughout the mesocolon, the result probably of old-standing infection from the dilated bowel.

Clinically, therefore, it would appear that megacolon may be due to a relative sympathetic overaction. In cases of megacolon no anatomical alteration is found in the extrinsic nerves of the colon, and we have ourselves dissected the parasympathetic sacral nerves passing to the lower bowel of a 6-months-old child suffering from megacolon and found no abnormality in them. Histologically also in cases of megacolon little change can be observed in the cells and fibres of Auerbach's plexus, so that the lesion would appear to be functional rather than organic.

ANATOMY AND PHYSIOLOGY.

Anatomy.—For a proper understanding of the experimental work which follows, it is advisable to give consideration to the anatomy of those nerves that supply the pelvic viscera—the sympathetic and the parasympathetic. We shall deal only with the efferent fibres, first with those passing to blood-vessels in general, and secondly with those passing to the musculature of the viscera.

Sympathetic nerves leave the cord in common with the anterior roots of the spinal nerves from the 2nd thoracic to the 3rd lumbar inclusive. They arise from cells in the intermedio-lateral horn and their function is essentially motor. The sympathetic nerves leave the spinal nerves and pass to the appropriate ganglia of the sympathetic ganglionic chain as white rami communicantes. These fibres are medullated and constitute the pre-ganglionic fibres. Within the ganglion some of these fibres form a synapse around a nerve-cell and thence there arises a second set of fibres. These fibres are non-medullated and constitute the post-ganglionic fibres. These pass to the

appropriate spinal nerve as a gray ramus communicans and are distributed to the blood-vessels along with the branches of the spinal nerve.

Those sympathetic fibres that are destined for the various viscera take another course. They arise in the cord in the same way and pass to the lateral sympathetic ganglionic chain as white rami communicantes. They pass out from the ganglionic chain without a synapse and thus retain their essential character of pre-ganglionic fibres. Similar fibres from neighbouring ganglia tend to join up together and there is thus constituted a splanchnic nerve. The fibres in a splanchnic nerve end in a synapse in one of the peripheral ganglia, such as the cœliac ganglion, and thence the post-ganglionic fibres pass to supply the particular organ.

The sympathetic nerves for the rectum are collected in the hypogastric or presacral nerve of Latarjet,[20] which they reach through the middle and the two lateral (splanchnic) roots of that nerve (*Figs. 85, 86*). The middle root is the continuation downwards of the aortic or intermesenteric plexus, which has intimate connections above with the cœliac, the renal, and the superior mesenteric plexuses. In these plexuses the majority of the fibres of the middle root have their cell station. These fibres remain intact after the operation of lumbar ganglionectomy and their division is one of the advantages of the 'presacral operation' (Learmonth).

The lateral roots of the hypogastric nerve (the least splanchnic nerves) arise variably from the ganglia of the lumbar sympathetic chain, and pass forwards and medially to join with the lower extremity of the aortic plexus and form, at the level of the bifurcation of the aorta, the hypogastric nerve (*Figs. 85, 86*). The right lateral root has a connection of its own with the cœliac and both renal plexuses, while the left lateral root has a similar connection with the cœliac and the left renal plexuses (Learmonth[21]).

The hypogastric (presacral) nerve enters the pelvis and then splits into two main branches which pass round the side wall of the pelvis, each to end in the hypogastric ganglion of its own side, which lies on the lateral wall of the rectum. In these ganglia the remaining pre-ganglionic fibres of the hypogastric nerve have their cell station.

The sympathetic nerves for the pelvic colon leave the aortic plexus above the origin of the hypogastric nerve and pass outwards to be distributed with the branches of the inferior mesenteric artery. The lowest of these nerves (the superior hæmorrhoidal nerve) accompanies the artery of that name to end in the hypogastric ganglia.

The parasympathetic nerve-supply to the pelvic viscera arises as white rami communicantes from the 2nd, 3rd, and 4th sacral nerves. These nerves tend to join up to form one nerve-trunk which is known as the nervus erigens or pelvic nerve. This nerve passes through the hypogastric ganglion to have its branches distributed to the various pelvic viscera in common with the post-ganglionic sympathetic fibres. The parasympathetic fibres end in ganglia in the walls of the viscera they supply, and from these ganglia there pass very short post-ganglionic fibres to the muscles of the viscera.

On the hypogastric ganglion, therefore, converge the autonomic nerves for the pelvic viscera. The nerves diverging from the ganglion are mixed nerves composed of sympathetic and parasympathetic fibres.

Physiology.—Stimulation of the sympathetic nerves causes a contraction of the sphincters of the bladder and rectum and a dilatation of the walls of these viscera. Stimulation of the parasympathetic nerves causes an opposite effect—a dilatation of the sphincters and a contraction of the walls, to allow

FIG. 85.—Dissection of a human female full-time fœtus to show the autonomic nerves of the lower abdomen.

of emptying of the particular viscus. In health we imagine the two sets of nerves to be in a state of physiological balance, the emptying of a viscus being determined by a relative overaction of the parasympathetic nerves,

and filling of the viscus by a relative overaction of the sympathetic nerves. Between the longitudinal and circular muscle coats of the colon, as of the whole intestinal tract, lies Auerbach's plexus of nerve-cells and filaments, without which it would appear that peristalsis cannot occur. The plexus has connections with the extrinsic sympathetic and parasympathetic nerves and its fibres end in the muscle cells of both coats of the bowel. A similar plexus (Meissner's) lies in the submucosa and is concerned with secretion.
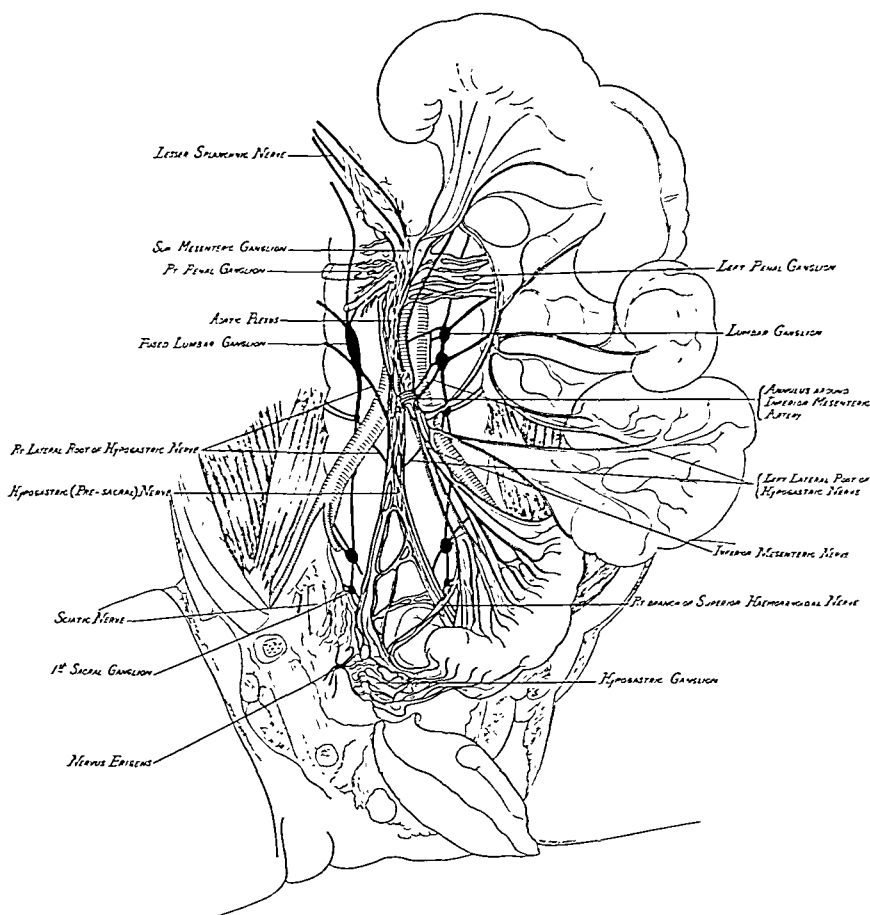
FIG. 86.—Autonomic nerves of the lower abdomen of a human female—a tracing of *Fig.* 85.

Anatomy of the Cat.—The anatomical arrangement of the autonomic nerves to the pelvic viscera in the cat differs only slightly from that found in man, in that there is a ganglion at the root of the inferior mesenteric artery (the inferior mesenteric ganglion) which serves as a cell station for the majority of the fibres of the hypogastric nerve. In this animal, therefore, the hypogastric ganglion is less cellular than in man.

EXPERIMENTAL MEGACOLON.

The aim of our work has been to produce experimentally a condition of megacolon of neurogenic origin. This could be achieved by upsetting the balance that exists between the parasympathetic and the sympathetic nerve-supply to the colon, so as to produce a relative overaction of the sympathetic nerves. This can be arrived at either by prolonged over-stimulation of the sympathetic nerves—a difficult thing to do experimentally—or by cutting out the action of the parasympathetic nerves. The latter course was chosen and the animals used were cats.

The cats were anæsthetized and, as a preliminary a barium enema was was given and a series of X-ray photographs taken. The barium, made
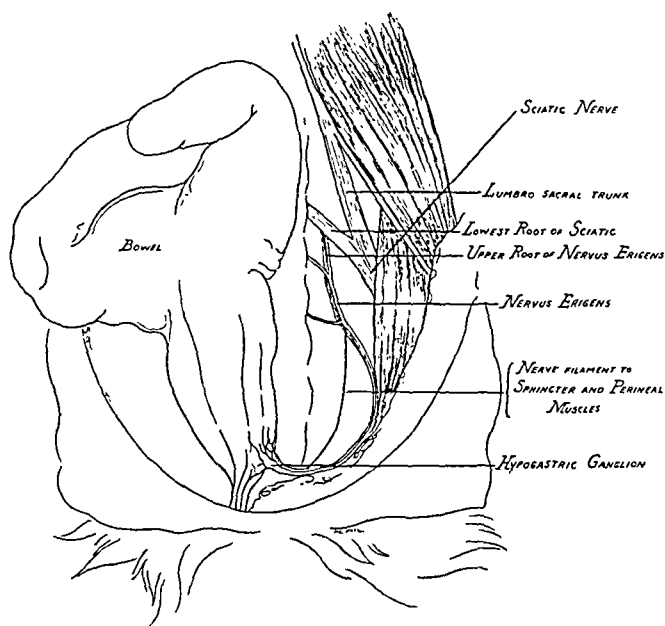
Fig. 87.—Arrangement of the nervus erigens in the cat.

up with water to the consistence of cream, was given through a No. 6 soft rubber catheter. To constitute a check on the quantity of barium injected it was decided to introduce the barium until a definite pressure was reached. After experiment it was found that a pressure of 55 mm. of mercury within the bowel was just sufficient to open the ileocæcal valve. With an enema given therefore at this pressure, we were confident of obtaining the X-ray photograph of a full colon. After the barium enema had been given, an X-ray photograph was taken and the animals were allowed to recover.

After a few days had been allowed for the animals to pass the barium, the operation for dividing the parasympathetic nerves was carried out. The abdomen was opened in the mid-line and the small intestine packed upwards.

The colon was pulled out of the abdomen and an incision made in the posterior peritoneum just below the bifurcation of the aorta, care being taken to avoid the presacral nerve. The edges of this incision were retracted and dissection carried down behind the rectum into the pelvis.

The landmark that was first displayed was the sciatic nerve. This was traced proximally, and passing from its lowest root downwards in a more vertical direction a small nerve—the uppermost root of the nervus erigens—was constantly found. The lower root or roots were then easily identified, and were traced proximally to their exit from the sacral foramina (*Fig.* 87). The arrangement of the roots of the nervus erigens, and indeed of the whole of the sacral plexus, varied considerably with the individual animal. In the majority the nerve had its origin by three roots, the uppermost of which was common to the lowest root of the sciatic nerve. In the operation the uppermost root was divided and the remaining roots were avulsed from the spinal canal, so as to minimize the possibility of regeneration. The trunk of the nervus erigens was itself divided proximal to the hypogastric ganglion, as large a segment as possible being thus resected. The procedure was carried out on both sides. The operation led to division of the parasympathetic nerve-supply to the colon and bladder.

The immediate effect of the operation was to cause the animals to suffer from retention of urine. Many methods were used in attempting to overcome the urinary difficulties. The employment of catheters, either permanent or intermittent, always led to infection of the bladder. Cystostomy, on similar lines to colostomy, with delay in opening the bladder till three days after operation, failed. Eventually it was found that opening of the bladder, stretching the bladder sphincter by the insertion of a pair of artery forceps through the bladder neck, and immediate closure of the bladder, achieved success and enabled the animals to live on indefinitely.

In addition to the division of the parasympathetic nerve-supply to the bladder and colon, the operation led to division of the voluntary nerve-supply to the external anal sphincter, the levator ani, and the perineal muscles, so that the sphincter became a flaccid ring and a mild degree of prolapse developed. The voluntary supply to these muscles usually ran in a small filament arising in common with the lowest root of the nervus erigens.

In all, 30 animals have been operated on. Of these, 25 have died at intervals after operation ranging from five to ten days. The cause of death in all these was renal failure. The post-mortem findings consistently showed a markedly dilated bladder, containing purulent urine which was often blood-stained. There was no dilatation of the ureters or of the renal pelves. The kidneys were congested and microscopic sections showed a marked degree of fatty degeneration with nephritic changes. These animals, therefore, died of renal failure from the backward pressure brought about by the loss of detrusor action of the bladder. The five animals that survived the operation continued to live in comparative comfort, and their bowels moved daily. At intervals after operation they were X-rayed after a barium enema, given at the standard pressure of 55 mm. of mercury. It was found constantly that after six weeks there was definite radiological evidence of dilatation of

the colon, and the quantity of barium held was increased.   By the end
of ten weeks the dilatation of the colon was well marked, and by the end of
fifteen weeks, the longest period for which we were able to allow an animal
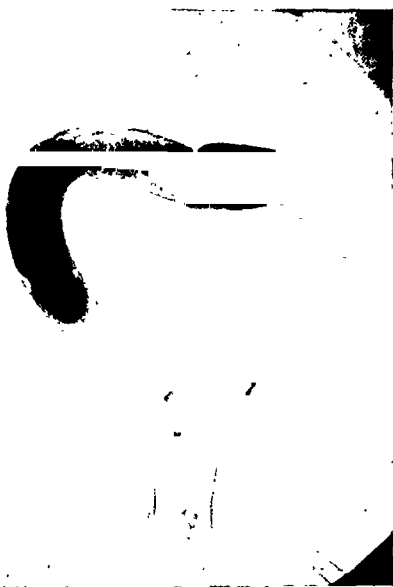
FIG. 88.—*Cat A.*   Pre-operative appearance.

FIG. 89.—*Cat A.*   Eight weeks after operation.   FIG. 90.—*Cat A.*   Twelve weeks after operation.
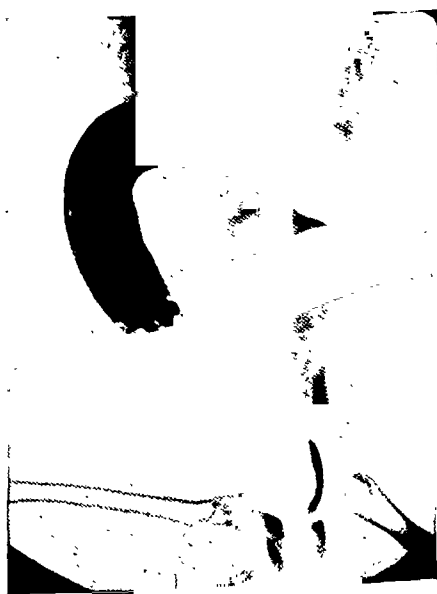
to survive, the dilatation was gross. The dilatation of the bowel was thus progressive. At death a post-mortem examination was carried out and the illustrations show the condition of the colon. Microscopic sections were

FIG. 91.—*Cat B.* Pre-operative appearance.

FIG. 92.—*Cat B.* Six weeks after operation.   FIG. 93.—*Cat B.* Fifteen weeks after operation.

made of the wall of the bowel, and these showed virtually no hypertrophy of the muscle coats. Furthermore, we were unable to satisfy ourselves that there were any changes in Auerbach's plexus. The urinary bladders in these cases showed very marked hypertrophy, with some dilatation. There was also some infection of the bladder, while the kidneys showed congestion of the pelves and fatty degeneration of their parenchyma. We ascribed the cause of death to the changes in the kidneys. The X-rays and the illustrations of the specimens are of two cats, the first (*Cat A*) having lived for twelve weeks after operation, and the second (*Cat B*) for fifteen weeks after operation (*Figs.* 88–95).

FIG. 94.—A, Colon of *Cat A* ; B, Colon of normal cat of same weight and size.
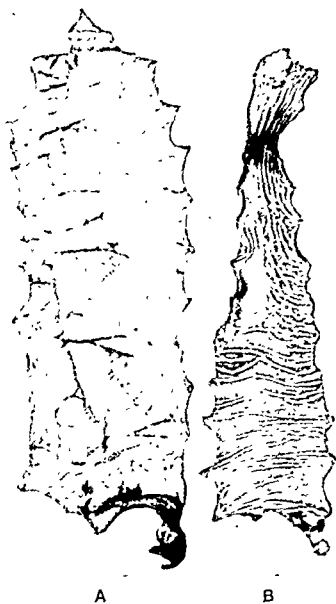
FIG. 95.—A, Colon of *Cat B.*   B, Colon of normal cat of same weight and size.

Thus we have succeeded in producing experimentally a megacolon, based on relative sympathetic overaction. The condition took eight weeks to become obvious, and thereafter was gradually progressive for so long as the animal lived. This experimental result agrees with the conclusion to which we have already been led by clinical observation.

While, therefore, we feel convinced that the condition is of neurogenic origin, the exact cause of the sympathetic-parasympathetic imbalance must remain obscure until we learn more of sympathetic-parasympathetic balance. Our knowledge of the control exerted by the central nervous centres on the bowel through the medium of Auerbach's plexus, of the modification of that control by the glands of internal secretion (for megacolon occurs in 30 per cent of cases of acromegaly) and of the relation of the nervous system to growth, hypertrophy, and gigantism, is still too slender to serve as a basis for a complete theory of megacolon.

SYMPATHECTOMY FOR MEGACOLON.

The essential points in the operation of sympathectomy for megacolon as recommended by Learmonth are clear from the illustration (*Fig.* 96). It is necessary that all the sympathetic fibres to the rectum and pelvic colon be divided.

After the abdomen has been opened and the small intestine packed upwards and to the right, and the large intestine either drawn downwards or out of the abdomen, the peritoneum over the bifurcation of the aorta is incised in a vertical direction for about 3 in. By a process of blunt dissection
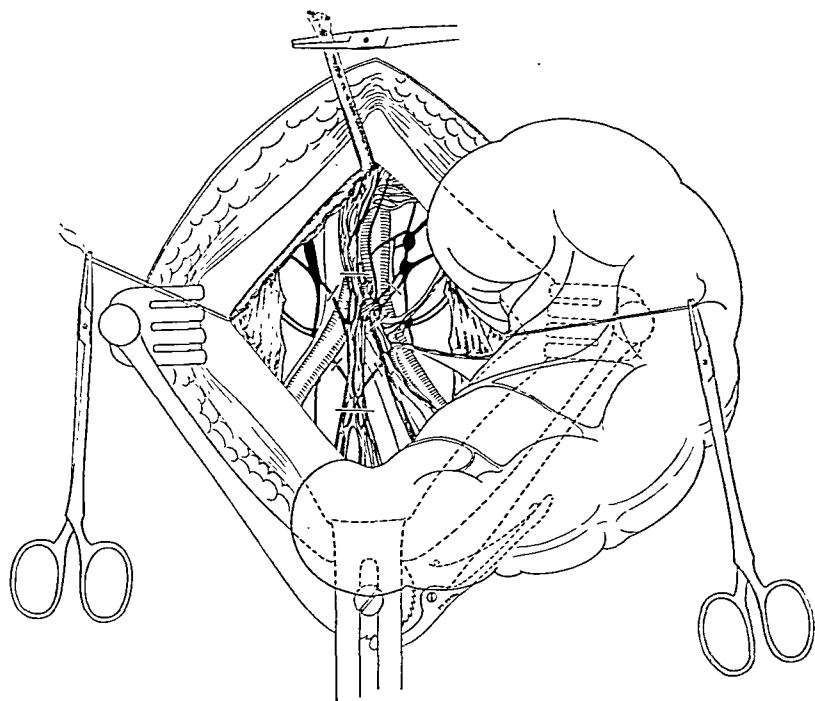
FIG. 96.—Situations at which the sympathetic nerves of the lower colon are to be divided in the operation for megacolon.

the bundle of nerve-fibres constituting the hypogastric or presacral nerve is easily found lying in the retroperitoneal tissues. The hypogastric nerve is followed downwards for about 1 in. and is then divided across between artery forceps. The forceps on the upper cut end of the nerve then acts as an efficient retractor. By drawing the nerve over to the patient's left side, the root from the 4th right lumbar ganglion becomes evident passing either in front or behind the common iliac vessels. This is divided, and the roots from the 3rd, 2nd, and 1st lumbar ganglia are divided as they come into view. These may be separate connections or in the form of a single lateral root. By further drawing on the nerve in a downward direction the middle root

from the aortic plexus is made to stand out and is divided. The left root of the nerve is then displayed and the connection from the 4th left lumbar ganglion divided. The left root is traced upwards and is found either to pass in front of, or behind, or as an annulus around the root of the inferior mesenteric artery. The commonest arrangement is the last. The anterior loop around the artery is divided (*Fig.* 97) and this permits of the whole
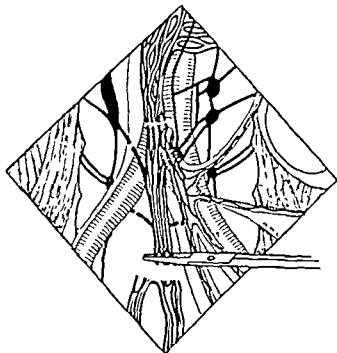
FIG. 97.—Division of the right lateral root and the middle root of the hypogastric nerve. The annulus around the inferior mesenteric artery has also been slit up.
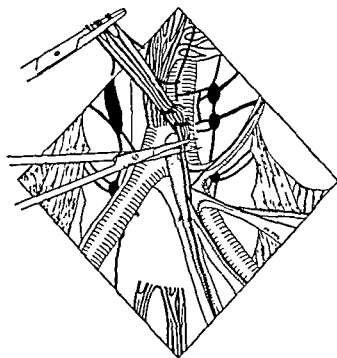
FIG. 98.—Manner in which the whole of that part of the hypogastric nerve to be removed is swung up behind the inferior mesenteric artery. The inferior mesenteric nerves are about to be divided, and the left lateral root of the hypogastric nerve will be divided at the points marked.

nerve. being passed upwards and to the left behind the artery and then drawn to the patient's right above and in front of the artery. By this manœuvre the nerves passing along the inferior mesenteric artery are made to stand out and these are all divided (*Fig.* 98). The left lateral root, by which alone the nerve now remains attached, is divided and the segment of nerve removed. The wound in the posterior peritoneum is sutured and the abdomen closed.

SUMMARY.

1. Clinical evidence is brought forward in support of the view that megacolon has a neurogenic origin.

2. Experimentally it is shown that a gradually progressive megacolon can be produced in the cat by removal of the parasympathetic nerve-supply to the distal colon.

3. A description of the autonomic nerves to the distal colon in the human subject is given, with the operative procedure recommended in cases of megacolon.

of megacolon. We have had valued help from Sir Arthur Keith in drawing our conclusions. and from Dr. F. E. Reynolds in the field of neuro-histology, and to these we would tender our thanks. It only remains to mention Mr. Frank Pettigrew and the staff of the Surgery Research Department of the University of Edinburgh for their skilled assistance. The drawings are the work of Miss McLarty.

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THE END-RESULTS OF PERINEAL EXCISION AND OF RADIUM IN THE TREATMENT OF CANCER OF THE RECTUM.

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IN the following study a survey is made of 370 cases of rectal cancer in which perineal excision was performed in St. Mark's Hospital from 1910–31, and of 89 cases in which radium has been employed since the latter part of 1928.

PERINEAL EXCISION.

The Operation.—The operation has been that described by Lockhart-Mummery[1] in 1920. At a preliminary operation a left rectus or left iliac colostomy is established. The colostomy has in many cases been a 'blind' one, without any attempt at abdominal exploration; in other cases the abdomen has been explored through the rectus incision through which the colon was finally brought out, or it has been explored locally through a left oblique muscle-split. In a considerable number of cases the abdomen has been thoroughly examined through a paramedian incision, the colostomy being brought out through a separate left oblique incision, and, personally, I favour the last mentioned routine.

The perineal excision has in most cases been performed seven to fourteen days after the colostomy operation. In the last ten years the operation has been done in the majority of cases under spinal anæsthesia, either combined with gas and oxygen, or in conjunction with preliminary narcosis by morphia and scopolamine, avertin per colostomy, or nembutal by mouth. The excision operation has been done with removal of the sphincters, the coccyx, and sometimes a portion of the sacrum. The peritoneum has been opened, the bowel freed and divided well above the growth, with as high ligation of the vascular pedicle as each case permitted. The bowel has been closed by suture and the peritoneal floor repaired.

Operability Rate of Cancer of Rectum.—Before describing the immediate and remote results of perineal excision, the following tables are recorded showing the operability rate in St. Mark's Hospital. The twenty-two years 1910–31 inclusive are divided into two periods of eleven years each, and the way in which the rate has been estimated is clearly shown by recording the different types of treatment that have been given; a summary of the hospital practice in relation to rectal cancer is thus obtained (Tables I, II). It will be noted that the operability rate has risen from 41·6 per cent in the first period to 54 per cent in the last eleven years.

The Cancer Follow-up Scheme at St. Mark's Hospital has rendered possible the compilation of these tables, as every case of cancer admitted

into the hospital has been included in the scheme and has been cross-refer-enced according to the treatment given. The tables are self-explanatory and only certain items need elaboration.

Table I.—Operability Rate, 1910–20.

Operable [o]		Inoperable	
Perineal excision	78	Palliative colostomy	187
Abdomino-perineal excision ..	37	Exploratory laparotomy ..	6
Abdomino-anal excision ..	8	Expectant	24
Perineal excision with preserva-			
tion of anus	4		
Perineal excision with sacral anus	11		
Hartmann's operation.. ..	1		
Local resection	5		
Expectant (refused operation) ..	11		
Total	155	Total	217

[o] In 372 cases 155 were operable = 41·6 per cent.

Table II.—Operability Rate, 1921–31.

Operable [o]		Inoperable	
Perineal excision	292	Palliative colostomy, alone or in	
Abdomino-perineal excision ..	39	conjunction with radium ..	281
Perineo-abdominal excision ..	10	Exploratory laparotomy ..	3
Hartmann's operation ..	3	Radium without colostomy ..	14
Local resection	7	Expectant	24
Radium	16		
Expectant (refused operation)..	9		
Total	376	Total	322

[o] In 698 cases 376 were operable = 54 per cent.

1. The cases classified as local resections were either early operable cancers which were removed *per vias naturales* or by posterior proctotomy, or they were villous tumours or adenomata which were removed by the same routes and were proved on section to have undergone malignant degeneration.

2. Squamous carcinoma of the anal canal and anus is included in esti-mating the operability rate, and accounts for 8 of the operable cases shown in *Table II* as having been treated with radium.

3. No case is included twice in these tables—for instance, cases of colos-tomy followed by perineal excision are shown under perineal excision only ; cases treated by radium followed later by perineal excision are similarly grouped under perineal excision only.

4. The radium cases have been assessed as operable or inoperable as accurately as possible, and grouped accordingly.

5. Inoperable cases grouped under palliative colostomy represent a small

error, for contained in this group there are some cases of inoperable carcinoma of the colon. It has not been possible to go through the entire series of case cards to separate the colon cases, and on this account the operability rate, so far as the rectum is concerned, should really be slightly higher than the figures as now quoted.

Operation Mortality of Perineal Excision.—The following collected figures from St. Mark's Hospital summarize the results of a group of operators and provide an accurate record of the progress that has been made with this operation in the course of the last twenty-one years. The operation mortality will be set out in three ways : (1) In relation to increasing experience with the operation ; (2) In relation to age and sex ; (3) In relation to the stage of the disease. In these tables operation mortality includes every case that died within four weeks after operation.

Increasing Experience with the Operation.—Examination of *Table III* shows that there has been a progressive decrease in the operative death-rate from 16 per cent in the first 100 cases to 10 per cent in the third 100 cases, and there is a good prospect that when the fourth 100 cases have been completed the mortality will prove to be down to a single figure. The mean operative mortality at present works out to 11·6 per cent (43 out of 370 cases). These results are a testimony to the keenness, co-operation, and

Table III.—OPERATIVE MORTALITY OF PERINEAL EXCISION IN RELATION TO INCREASING EXPERIENCE WITH THE OPERATION.

	M.	F.	DATE	OPERATIVE MORTALITY	M.	F.
First 100 cases ..	68	32	1910–22	16	11	5
Second 100 ,, ..	66	34	1922–27	13	11	2
Third 100 ,, ..	68	32	1927–29	10	8	2
Next 70 ,, . ..	47	23	1930–31	4	3	1
Total 370 ,, ..	249	121	1910–31	43	33	10

good team work of everyone who is concerned with the cases. Zealous preparation and after-care, together with the extensive use of spinal anæsthesia, have been important factors. The decrease in the death-rate among males in recent years is noteworthy, and is, in my opinion, largely due to the interest which Dr. Dukes has stimulated in the subject of urinary infections and their prevention. His intensive campaign on this question and the evolving of his retained catheter apparatus dates from 1927–8. Its influence is clearly shown in this table in the decrease in actual male mortality from 16·2 per cent in the first 100 to 11·7 per cent in the third 100 cases. The table also shows the greatly increased number of these operations that are being done at the hospital ; whereas it took twelve years to complete the first 100 cases, the fourth ·100 will probably be completed in three years or less.

Age and Sex.—It will be seen from a study of *Table IV* that male cases represent slightly more than two-thirds of the total, with a mortality of 13·25 per cent (33 out of 249), the female mortality being 8·3 per cent (10 out of 121).

Table IV.—OPERATIVE MORTALITY IN RELATION TO AGE AND SEX, IN 370 CASES OF PERINEAL EXCISION (1910–31 INCLUSIVE).

AGE INCIDENCE IN YEARS	TOTAL NUMBER OPERATED UPON		NUMBER OF OPERATIVE DEATHS	
	Males	Females	Males	Females
20–29	4	2	1	—
30–39	10	11	1	—
40–49	49	21	3	2
50–59	94	42	12	2
60–69	80	38	11	5
70–79	12	7	5	1
Total	249	121	33	10

As regards age, 137 out of the total of 370 were subjects over 60 years of age, and the fact that in this series 19 patients over 70 years old have been operated upon shows that advanced age has not in itself been considered a bar to perineal excision. The heavier mortality in males over 70 years of age (5 deaths out of 12) is to be expected and was actually caused in this series by the following conditions : sepsis, cardiac failure, uræmia, exhaustion, and bronchopneumonia.

Operative Mortality in Relation to the Stage of the Disease.—The method of pathological classification of rectal cancers according to depth of spread has been described by Dukes.[2, 3] Briefly, his method is to incise the specimen, generally along the middle-line anteriorly ; the specimen is then pinned out to keep it flat and stretched. Fixation is effected in formol-saline and the measurements are taken of the length of the specimen, the size, situation, and extent of the tumour, together with the amount of free margin of bowel above and below. The growth is then sectioned through what appears to be the point of maximum penetration of the bowel wall by the growth, or at the point of deepest ulceration. Any enlarged lymph-nodes in the meso-rectum are identified by a series of thin transverse slices. Suitable blocks of the growth and lymphatic territory are prepared and sectioned : examination of the sections under a low power of the microscope enables the cases to be grouped in the following way :—

" *A* cases are those in which the carcinoma is limited to the wall of the rectum, there being no extension into the extrarectal tissues and no metastases in the lymph-nodes.

" *B* cases are those in which the carcinoma has spread by direct

continuity to the extrarectal tissues, but has not yet invaded the regional lymph-nodes.

" C cases are those in which metastases are present in the regional lymph-nodes." (Dukes.)

Table V shows that in A cases (32) the operative mortality was nil, in B cases (53) it was only 3·8 per cent, whereas in C cases (71) it was 12·7 per cent. This very instructive finding shows that a group of surgeons has been able to carry out 85 perineal excisions (groups A and B combined) with only 2 deaths (2·4 per cent), and suggests that once the necessary skill and experience have been obtained the mortality depends almost entirely upon the number of advanced C cases that are tackled by radical operation.

Table V.—Operative Mortality in Relation to Stage of Disease. Classification by Dukes' Method according to Depth of Spread (1927–31).

CLASSIFICATION OF GROWTH	NUMBER SUBMITTED TO OPERATION	DIED FROM OPERATION	OPERATION MORTALITY
A	32	0	Per cent 0·0
B	53	2	3·8
C	71	9	12·7
Total	156	11	7·0

At St. Mark's Hospital the principle upon which excisions have been done has been that it is worth while to excise any rectal cancer that is not hopelessly fixed, provided that metastases to the liver have not occurred. A deeply ulcerated growth with metastases in the glands may be just capable

Table VI.—Immediate Causes of Operative Deaths.

IMMEDIATE CAUSE OF DEATH	NUMBER	MALES	FEMALES
Heart failure, shock, hæmorrhage and exhaustion	16	12	4
Sepsis	14	11	3
Urinary complications	5	4	1
Pulmonary complications	3	3	0
Intestinal obstruction	3	2	1
Pulmonary embolism	2	1	1
Total	43	33	10

of removal before it has irremediably penetrated into the prostate or bladder base ; if the operation mortality is under 15 per cent. such an operation would appear to be worth while, and even if recurrence in the abdominal glands or liver should present itself within three years. the local removal of the growth will be justified by the freedom from pain and from local complications, such as rectovesical or rectovaginal fistulæ. which might otherwise have developed.

The Immediate Causes of Operative Deaths.—*Table VI* shows the causes of the 43 operative deaths in this series.

It will be observed that, contrary to the usual findings. 'sepsis' takes second place in the list of fatal complications. An unfortunate series of 4 fatal secondary hæmorrhages and 1 fatal reactionary hæmorrhage. probably due to an alteration in the technique of ligation of the superior hæmorrhoidal pedicle, puts the vascular complications at the head of the list. The small number of fatal chest complications, amounting to less than 1 per cent of the operations performed, is noteworthy. The 3 cases of intestinal obstruction were due to small-gut obstruction round the colostomy (2) and obstruction through the pelvic floor (1).

Late Results of Perineal Excision.—The late results of perineal excision are shown in *Tables VII* and *VIII*. drawn up on the generally accepted lines to show ten-year and five-year cures. and also in *Table IX*, which shows three-year cures grouped according to the proved depth of spread (groups *A*, *B*, and *C*).

Table VII.—RESULTS OF PERINEAL EXCISION AT 10 YEARS.

Total number of cases operated upon, 1910–21 inclusive 91	
Operative deaths 14	
Survivals from operation 77	
Alive and well at present time (1932) .. .. .. 17 }20	
Survived ten years and died from other causes .. .. 3 }20	
Died from recurrence .. .. 36 }47	
Died from cause unknown .. .. 11 }47	
Died from other causes in less than ten years .. .. 8 }10	
Untraced .. .. 2 }10	

If we subtract the 10 cases who are untraced or died from other causes in less than the ten years from the 77 survivals from operation, we obtain the figure of 67 cases, of whom 20 have been cured on a ten-year basis, i.e., 30 per cent.

Table VII may be elaborated by supplying the following facts : The longest-living survivor is a man upon whom Mr. Swinford Edwards operated in December, 1911—over twenty years ago. Colostomy and perineal excision were performed, the patient's age being 33. He has recently been re-examined ; he is in good health and has a good colostomy, the perineal wound is soundly healed, and there is no sign of recurrence. The period of survival of the other 16 cases is 17 years, 15 years (4 cases), 14 years (3 cases), 13 years (4 cases), 12 years, and 11 years (3 cases). Three patients died from other causes 15. 12, and 12 years after operation. No patient died of recurrence who survived the ten-year period. The small number of untraced cases (2) is noteworthy : one of these patients went to Australia three years after

operation and has not been heard of since, and the other, operated upon in 1920, has never been traced.

Table VIII.—Results of Perineal Excision at 5 Years.

Total number of cases operated upon, 1910–26	..	.. 189
Operative deaths	..	.. 26
Survivals from operation	..	.. 163
Alive and well at present time (1932)	..	45 ⎫
Survived five years and died from other causes	..	10 ⎭ 55
Survived five years and died from cancer ..	..	6 ⎫
Alive after five years with recurrence	..	2 ⎭ 8
Died from recurrence in less than five years ..	..	68 ⎫
Died from cause unknown	..	20 ⎭ 88
Died from other causes in less than five years ..	..	10 ⎫
Untraced	..	2 ⎭ 12

Table VIII is self-explanatory: if the 'untraced' and 'died from other causes' are subtracted from the operation survivals, 36·4 per cent of five-year cures are revealed (55 out of 151). Special mention should be made of the 6 cases that survived the five-year period and later died from cancer. Their deaths occurred as follows :—

5 years and 2 months	..	Growth in right lung, probably independent.
5 ,, ,, 7 ,,	..	Recurrence in blind segment of colon below colostomy.
5 ,, ,, 9 ,,	..	Recurrence in vagina and pelvis.
6 ,, ,, 7 ,,	..	Recurrence in blind segment of colon below colostomy.
7 ,, ,, 5 .,	..	Recurrence in blind segment of colon below colostomy.
9 ,, ,, 2 .,	..	Growth in right lung and in glands of neck.

The 3 cases in which recurrence took place in the bowel below the colostomy are noteworthy; these were probably carcinomas arising independently in pre-existing adenomata, and indicate the importance of removing as great a length of bowel at operation as possible. If these 3 cases, together with the 2 cases dying after five years of cancer of the lung without evidence of local or abdominal recurrence, are reckoned also as five-year cures, the figure for five-year cures becomes 40 per cent (60 out of 151).

Table IX.—Results of Perineal Excision at Three Years Classified according to Proved Depth of Spread of the Growth.

CLASSIFICA-TION OF GROWTH	NUMBER OF CASES (1927–March 1929)	DIED FROM OPERATION	DIED UNDER 3 YEARS		UNTRACED	ALIVE AT 3 YEARS	PERCENTAGE OF 3-YEAR CURES
			Of other Causes	Of Cancer			
A	15	0	1	2	—	12	86
B	24	1	—	6	1	16	73
C	21	4	1	13	—	3	19
Total	60	5	2	21	1	31	60

In *Table IX* the percentage of three-year cures after perineal excision is shown, with the cases classified according to the proved depth of spread. The percentage of cures is worked out from the usual fraction—

$$\frac{\text{alive at three years} \times 100}{\text{number of operation survivals less those untraced and died from other causes}}$$

It will be seen that in *A* cases there is the remarkable figure of 86 per cent of three-year cures, in *B* cases the percentage is 73, whereas in *C* cases it drops to 19. The difference in the prognosis of the three groups is thus strikingly indicated, and is likely to be more marked when time has elapsed for a similar table on a five-year basis to be drawn up.

RADIUM TREATMENT.

The treatment of rectal cancer by radium needle barrage has been carried out in St. Mark's Hospital since the latter part of 1928. The methods employed have been those described by Neuman and Coryn,[4] and by Sir Charles Gordon-Watson.[5, 6, 7] Minute details of technique need not be described here, and it will suffice to state that :—

1. An ample supply of radium has been available in amounts of 0·5, 1, 2, and 3 mgrm. per needle.

2. The needles have varied in length from 1·9 cm. to 6 cm.

3. The filtration has been 0·5, 0·6. and 0·8 mm. of platinum.

4. In placing the radium needles every effort has been made to obtain an even and well-distributed barrage.

5. In estimating dosage the principle has been to give as heavy a dose as seemed reasonable for each particular case. The average duration of the radium exposure has been six to eight days.

6. Radon seeds have been used in a few of the more recent cases.

The following report covers the complete series of 89 cases of rectal cancer that have been treated with radium in St. Mark's Hospital from 1928–31 inclusive. In some of the cases the radium treatment has been carried out from more than one aspect—for instance, an original abdominal or vaginal barrage has sometimes been followed by intrarectal insertion of radium needles or radon seeds, but the principal attack has been the first one and the cases have been classified for the sake of clearness in the following way :—

		CASES
I. *Carcinoma and Sarcoma of the Rectum—*		
1. Operable cases treated with radium		14
2. Radium in conjunction with perineal excision		11
3. Abdominal radiation		16
4. Posterior barrage		9
5. Interstitial needling for inoperable cancers		18
6. Recurrent carcinoma		9
II. *Squamous Carcinoma of the Anus and Anal Canal*		12
Total		89

I. CARCINOMA OF THE RECTUM.

1. **Operable Carcinomas Treated with Radium.**—Fourteen operable carcinomas have been treated with radium : there were 12 females and 2 males. The cases fall into two groups : (*a*) 12 cases treated with radium needles; (*b*) 2 cases treated with radon seeds.

a. Radium needles were inserted from·the vagina, perineum, or peri-anal skin in 12 cases, with the following results : 2 cases are alive and well for periods of 3 years 2 months (with colostomy) and 2 years 10 months (without colostomy) after the operation. The following are the details of these two cases :—

Case 1.—Female, age 60. Movable fungating tumour in lower third of rectum, situated anteriorly and on the left side. October, 1928—31 radium needles, total 22·1 mgrm., inserted for 68 hours ; dosage 1502 mgrm. hours. November, 1928— left inguinal colostomy. December, 1928—second radiation carried out, 5 needles totalling 12 mgrm. being inserted from outside the anus for 152 hours ; dosage 1824 mgrm.-hours. April, 1929—third radiation, partly via lumen of bowel, partly from outside ; dosage 2688 mgrm.-hours. July, 1929—re-admitted for treatment of radium burn of anal canal and removal of a slough from the left ischiorectal fossa. The patient made a gradual recovery, and in December, 1931, reported that her health was good and that she had no symptoms of recurrence.

Case 2.—Female, age 61, with a small, hard malignant tumour on the right lateral wall of the lower rectum. The diagnosis of adenocarcinoma was proved by microscopical examination of portions of the growth removed with Brünings' forceps. The patient was a stout bronchitic subject unsuitable for radical operation. In May, 1929, interstitial radium needling was carried out by separate punctures external to the anus and per vaginam—total 26 mgrm. for 10 days ; dosage 6240 mgrm.-hours. Colostomy was not performed. The new growth retrogressed very rapidly. The patient is now in good health and free from rectal symptoms ; her bowels act normally. The site of the original tumour in the rectum is marked by a smooth, depressed scar without any outlying induration. Abdominal examination is negative.

Three cases are alive, but with symptoms of recurrence, 2 years 2 months, 2 years, and 1½ years (all without colostomy). One case died with pleurisy, jaundice, and ascites three months after interstitial needling of an operable adenocarcinoma.

Six cases subsequently required perineal excision for the following reasons : (i) Three for lack of response to radium ; one of these cases had two further applications of radium or radon before excision was decided upon, and one case had one further radium needling. (ii) One on account of finding a second growth higher up. (iii) Two for recurrence of the growth locally ; the excision operation in these 2 cases being done twenty-one months after the initial radium treatment (both proved to be C cases). Four out of the 6 perineal excision cases are now alive and well, 2 years 3 months, 2 years 3 months, 6 months, and 3 months after operation. One is dead from secondary deposits in the lungs, and one died from mediastinal growth.

b. Two recent cases were treated by intrarectal radon seeds, one after colostomy and one after local excision of a small rectal cancer. Both are at present alive and well, the tumours having disappeared.

Commentary.—It is seen therefore that out of 12 operable cases treated with radium needles as the primary method of treatment there are only 2 good results achieved by radium alone. The fact that such results are capable of being achieved is important ; it would be more useful still to know why these cases responded well and how such good results could be repeated. In 6 of the cases a trial of radium treatment had to be terminated by perineal excision and the loss of valuable time in these cases is evident. Attention should be directed to three of the bad results in this series of 12 cases.

i. Death from pleurisy and jaundice three months after interstitial needling of an early growth.

ii. Death from secondary deposits in the lungs fifteen months after perineal excision and nineteen months after the original interstitial radium needling per vaginam. The operation specimen was classified as a *B* case.

iii. Death from a mediastinal growth thirteen months after perineal excision. This case had previously received two treatments by radium needling nineteen and seventeen months before death.

The conclusion is almost forced upon one that these unusual results can only be explained by vascular embolism set up by the radium needling.

2. Radium in Conjunction with Perineal Excision.—In 11 cases treated by perineal excision, radium has been employed also, the indications being a suspected extension of the growth beyond the limits of the operation. The radium needles have been inserted into the upper part of the mesorectum, along the lateral ligaments, and into the base of the prostate.

There was one operative death on the tenth day ; post-mortem examination showed a pelvic peritonitis to be present without any definite evidence of the source of the infection.

Of the 10 operative survivals, 5 were classified as *B* cases, 5 as *C* cases. One *B* case died three months after operation, probably from urinary failure ; 3 *B* cases are alive and well for periods of 3 years, 3 years, and $1\frac{1}{2}$ years after operation ; and 1 *B* case was alive and well at a year after the operation, and since then he has not been traced, as his work has taken him away from his home. Of the 5 *C* cases, 4 are dead from recurrence in less than 18 months after operation ; 1 is alive 18 months after operation—his health is poor, and he is stated to have secondary malignant deposits in the iliac glands.

Commentary.—The conclusion is that in *B* cases radiation is unnecessary, and in *C* cases there is no evidence that the radium treatment was beneficial either in preventing recurrence or in otherwise assisting the treatment by operation.

3. Abdominal Radiation.—Abdominal radiation by radium needles or radon seeds has been employed in 16 cases : there were 15 cases of carcinoma and 1 case of sarcoma.

a. In 13 cases the patients had inoperable cancers of the rectosigmoidal junction. In 2 cases colostomy had been performed previously, in 1 case colostomy was performed when the radium was removed on the seventh day, in 1 case colostomy was not done, and in the remaining 9 cases colostomy was performed at the time of insertion of radium needles or radon. Operative death occurred in 6 cases ; there were 4 deaths from peritonitis from the third to the eighth day after operation, 2 of these cases having

required also a resection of small intestine ; 1 death occurred on the second day from intraperitoneal hæmorrhage, and 1 death on the twenty-fourth day from gradual failure. Of the 7 cases that survived operation, 5 died unrelieved at periods varying from 3 months to a maximum of 18 months after operation, and the following are the details of two of these cases :—

One case, a male, age 59, with an inoperable growth in the upper third of the rectum, was treated by colostomy and insertion of radon seeds intraperitoneally into the growth. Later, radon seeds were inserted into the growth intrarectally on two occasions, and fourteen months after the initial treatment sigmoidoscopy showed a smooth cavity, lined apparently by normal mucosa, without any sign of carcinoma locally. The patient, however, died eighteen months after the original operation, probably with secondary deposits in the liver.

One patient with a large inoperable growth in the upper third of the rectum was treated by colostomy and insertion of 22 mgrm. of radium, the needles being passed along catheters into and around the growth. Prolonged suppuration in the abdominal wall followed, and finally cystoscopy proved the growth to be infiltrating into the base of the bladder ; the patient died unrelieved three months after operation.

The following are the details of the two cases that are still alive after abdominal radiation for inoperable cancers in the upper one-third of the rectum :—

Case 1.—Male, age 70, with a fixed carcinoma in the upper one-third of the rectum was treated in April, 1929, by colostomy and intraperitoneal insertion of 11 mgrm. of radium. Five weeks later sigmoidoscopy showed a marked radioreaction, with diminution in size of the growth with less hardness and fixity. In November, 1929, five radium needles (each 2 mgrm.) were inserted into the growth intrarectally. In December, 1931, i.e., more than two and a half years after the original operation, he reported himself as being in good health and able to do gardening.

Case 2.—Male, age 47, with an inoperable growth in the upper third of the rectum was treated in August, 1929, by colostomy and intra-abdominal radium. Later, radon seeds were inserted per sigmoidoscope twice. In December, 1931, he was reported to be alive, but in very poor health, with recurrent symptoms.

b. The three remaining cases fall into different categories :—

In one, a female, age 58, a perineal excision of the rectum was performed on account of an ulcerated tumour in the lower third of the rectum, with massive, upward lymphatic spread. Section proved the growth to be a round-celled sarcoma. Six weeks later laparotomy was performed and 17 mgrm. of radium were inserted into the root of the pelvic mesocolon for ten days. Three months later a perineal recurrence was treated by interstitial needling. The patient died from the disease seven months after the excision operation, a rapidly growing subcutaneous nodule in the back proving that vascular embolism had taken place.

One case, a male, age 62, had a pedunculated malignant adenoma, the size of a hazel nut, avulsed per sigmoidoscope from the rectosigmoidal junction, accompanied by intraperitoneal insertion of five 0·5-mgrm. radium needles into the related portion of the mesorectum for nine days. The

patient reported himself to be well and free from symptoms two and a half years later. There is no definite proof, however, that this very early carcinoma was not completely removed by the local excision.

One case, a female, age 61, had a carcinoma of the pelvic colon removed by Paul's operation, with insertion of radium down to the pouch of Douglas on account of definite subperitoneal nodules. Eleven months later multiple secondary deposits were present and the patient died seventeen days after a short-circuiting operation to relieve obstruction.

Commentary.—The intra-abdominal method of radium treatment for inoperable rectal cancers is shown by this series to be attended with a high operative risk, and the late results do not appear to justify the added risks when compared with the results of simple palliative colostomy.

4. **Posterior Barrage.**—Radium needling by posterior barrage has been carried out in 9 cases. In 3 the needling was done without a previous colostomy, in the remaining 6 a colostomy had either been established prior to the radium barrage or was effected at the same time.

Three patients died from post-operative toxæmia on the fourteenth, thirty-eighth, and forty-fifth days after operation. One patient died from recurrent secondary hæmorrhage from the perineal sinus three and a half months after operation. One had a severe hæmorrhage from the colostomy eight months after the operation and finally died from secondary growth two years after the radium insertion. One patient died nine months after operation, and one is untraced. The following are the details of the two cases that are known to be alive :—

Case 1.—Male, age 66, with a large rectal carcinoma adherent to the sacrum. Radium needling by posterior barrage was carried out in July, 1929 (38 mgrm. for 7 days = 6384 mgrm.-hours). Colostomy was not done. Rapid retrogression occurred, and in November, 1929, the rectum was smooth without any sign of tumour formation. In December, 1931, he reported that he was in very good health and able to do office work.

Case 2.—Female, age 53, with a large, fixed ulcerated growth in lower third of the rectum. Oct. 3, 1930—left iliac colostomy. Oct. 21—posterior and anterior radium barrage (52 mgrm. for 7 days = 8736 mgrm.-hours). The patient made a slow convalescence and in March, 1931, she had gained weight, and the rectum presented a smooth stricture, without any sign of neoplasm. In February, 1932, the condition was unchanged, there being a marked fibrous stenosis of the rectum, free from ulceration.

Commentary.—There are thus 2 good results in a series of 9 cases. The dangers of the method are evident and are indicated by the fact that 3 of the cases required hospital treatment for more than three months after operation, on account of severe wound sepsis.

5. **Interstitial Needling for Inoperable Adenocarcinoma of the Rectum.**— Eighteen cases (8 males, 10 females) have been treated by interstitial needling from the perineum, peri-anal circumference, or vagina : 7 cases were treated without colostomy and 11 cases had a colostomy performed previous to operation.

There was one operative death from toxæmia on the 13th day (male, age 56). Six cases died unrelieved at periods varying from 5 weeks to 2 years after the radium treatment, and 1 other case at present untraced

probably died 2 years after operation. Four cases have been much relieved and are now free from rectal symptoms 14 months, 16 months, 18 months, 18 months since operation. Two of these cases have colostomy, and 2 were treated without colostomy. Four cases have been relieved, but still have rectal symptoms 8, 9, 15, and 19 months later, and 2 cases are recent.

Commentary.—This type of radium needling evidently has a smaller operative risk as compared with the posterior barrage operation, but even so several cases passed through a long period of post-operative suppuration, with necessity for incision of perirectal abscesses and slow removal of sloughs. Two females developed rectovaginal fistulæ.

6. Radium for Recurrent Carcinoma.—

a. Seven cases of local recurrence in the perineum, vaginal wall, or ischio-rectal fossa, occurring at varying times after perineal excision, have been treated by interstitial radium needling. Five of these cases have died unrelieved at intervals of 4, 5, 7, 8, and 15 months after the radium treatment. One case is alive 20 months after radium was first applied to a perineal recurrence. Radium has recently been re-inserted for the third time and tissue removed from the margins of the ulcer has been shown to contain deposits of adenocarcinoma which are well differentiated and actively proliferating. One case treated in October, 1931, is at present relieved.

b. Two other cases of recurrent carcinoma need separate mention.

Case 1.—Female, age 54, with a small operable carcinoma low down in the anterior rectal wall. In May, 1930, the growth was excised locally. One month later the scar was found to be slightly nodular, and the scar was irradiated by vaginal needling. In November, 1931, she was examined and found to be free from recurrence.

Case 2.—Female, age 55, with a mass of abdominal recurrence eight years after perineal excision of the rectum. In October, 1928, abdominal implantation of radium into the pelvic mesocolon was carried out. The patient *died in* February, 1930.

Commentary.—These results suggest that there is very little likelihood of an established recurrence being more than temporarily improved by interstitial needling.

II. SQUAMOUS CARCINOMA OF THE ANUS AND ANAL CANAL.

Twelve cases of this type have been treated by radium needles: of these 8 are assessed as 'operable', and 4 as 'inoperable'.

Operable Cases.—Of these 8 operable cases there have been 4 good results in which the anal carcinomas have completely disappeared as a result of the treatment, and colostomy has not been required. Three of the cases are alive and well at periods of 3 years, $2\frac{1}{2}$ years, and $2\frac{1}{2}$ years since operation, and the fourth case has recently died from bronchopneumonia 2 years after the treatment. His anal condition was subjected to careful examination shortly before his death and there was no sign of recurrence.

Of the remaining cases one was relieved for 6 months and then local recurrence became evident, with a very rapid extension to the groin, which

exploration proved to be inoperable: death occurred 10 months after the original radiation. One case was relieved for 6 months, when local recurrence occurred and an extensive carcinoma of the cervix uteri was found. She died about 15 months after the radium operation.

One case died unrelieved 3 months after operation. One case of carcinoma of the anal canal, treated by colostomy and interstitial needling, is recent; the growth has already retrogressed markedly.

Inoperable Cases.—Of the 4 inoperable cases treated, 2 have died unrelieved, each living for exactly one year. One patient with a squamous carcinoma of the rectovaginal septum was relieved for a year. Two further insertions of radium were done, with temporary relief, and she died finally 3 years and 2 months after coming under observation. One other case is probably alive but unrelieved.

SUMMARY.

1. The results of perineal excision of the rectum are shown, and the importance of Dukes' classification of rectal cancers is emphasized, for this method enables the early cases to be separated from the late cases. *Table IX* reveals the excellent results, both immediate and remote, that can be expected in Groups A and B, together with the bad prognosis in those cases in which metastasis has occurred to the regional lymph-nodes.

2. The feeling of uncertainty and disappointment which is felt in regard to radium in the treatment of rectal cancer is reflected in the following table showing the number of new cases submitted to radium treatment in the last four years.

YEAR					NEW CASES TREATED WITH RADIUM
1928 (part only)	..	..	..	..	5
1929	..	..	..	..	44
1930	..	..	..	..	31
1931	..	..	..	..	9

In operable adenocarcinoma there have been two good results out of 12 cases treated: this is a poor showing compared with the results of perineal excision and accounts for the present opinion that radical operation holds out the best chance of cure to the patient.

The dangers of abdominal radiation and of posterior barrage have been sufficiently indicated. The futility of needling C cases at operation, and cases of recurrent cancer, is also evident. There is no case in this series in which an inoperable rectal cancer has been rendered operable by the use of radium.

The intrarectal insertion of radon seeds carries the least operative risk of the various methods of radium needling, but whether this form of radiation can produce anything better than a temporary palliation it is not yet possible to say from a study of these cases.

The most hopeful results have been obtained in the case of operable squamous carcinoma of the anus: this class of case having given 50 per cent of good results up to three years. This type of growth appears to respond

much better to radium than adenocarcinoma, but it must be taken into account that the growths are much more accessible and the needling can therefore be carried out more effectively.

This report has been compiled from the Cancer Follow-up Scheme of St. Mark's Hospital, which has been carried on under my direction for the last ten years with the help of an annual grant from the Medical Research Council. I need hardly say that my colleagues have always given their ready co-operation with the scheme, and I feel it a great privilege to present this comprehensive report on the results of our united efforts. The section dealing with the results of radium treatment is published with the permission of the Medical Research Council.

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DELAYED HÆMORRHAGE FOLLOWING TRAUMATIC RUPTURE OF THE SPLEEN.*

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RUPTURE of the spleen, whether traumatic or spontaneous, the result of perforating wounds or of subcutaneous injury, is usually rapidly fatal unless immediate operation is performed. There is, however, considerable variability in the evolution of the clinical signs, owing to the character of the internal hæmorrhage which dominates the symptoms of rupture of the spleen. As Bland-Sutton pointed out, the patient may collapse as dramatically as when an aortic aneurysm has burst. Vincent reported many instances in which death supervened in this manner. Thus, the accident may be brought into the category of those better-known conditions capable of producing sudden death, and should always be borne in mind in the investigation of such an occurrence, particularly if injury is recorded as having been sustained in the region of the spleen. From the therapeutic standpoint such cases are not of much interest. On the other hand, after the initial injury the patient's condition may gradually become worse, with obscure abdominal symptoms over a period of hours, days, or weeks, until the fact of progressive internal hæmorrhage becomes manifest and the surgeon's aid is finally sought. Particular significance is attached to the cases in which the clinical course is more prolonged, because of the too-often-forgotten fact that between the initial injury to the spleen and the appearance of signs of internal hæmorrhage there is usually a period of 'symptomatic silence' termed by Baudet the 'latent period'. This usually lasts from a few hours to one or two days and may lull the patient and physician alike into a false sense of security which may be rudely shattered by the rapid collapse of the patient under the cumulative effect of the active, internal hæmorrhage. Much more rarely this latent period exists for days or weeks and the patient goes about his work in the belief that the primary injury is not of much significance. The latent period in such cases is due to the absolute cessation of all bleeding from the injured organ and to its efforts at repair. It is terminated by the abrupt renewal of profuse secondary hæmorrhage under the stress of a slight rise in blood-pressure and is attended by the same high mortality as primary rupture unless operative intervention is immediately undertaken.

The occurrence of a typical example of the foregoing syndrome at the Mayo Clinic stimulated me to search for other examples, and, in view of the fact that adequate study of such cases has not appeared in English medical literature, I have collected and studied forty-six reports of cases from the

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literature, including the case which has been published in abstract by Counseller and me.[46] This case is reported in detail herein. It should be made clear that these represent examples of subcutaneous injury in which it was certain that primary rupture was followed by temporarily complete or almost complete cessation of hæmorrhage for a longer or shorter period, and this, by delayed bleeding of great severity. Since the latent period undoubtedly existed in cases in which hæmorrhage was still occurring I arbitrarily chose only those in which it lasted more than forty-eight hours and in which the clinical course undoubtedly indicated renewed secondary hæmorrhage. Furthermore, I have not included cases in which the patient gradually sank under the influence of a slowly progressive hæmorrhage and in which there was complete absence of the sudden collapse so typical of the particular syndrome I am describing. For this reason certain cases (d'Auvray,[3] Balloch,[5] Baudet,[7] Fiolle,[27] Hoffmann,[35] Metcalfe,[49] Neck,[51] Novis,[53] Page,[54] Rammstedt,[61] Stowasser[68]), including the second case of Flammer, could not be accepted in the series that I am presenting in review, although foreign authors have included them in their tabulations. Six cases were included in which the details were meagre but no doubt existed as to their nature (Bland-Sutton,[11] Freund-Kirchner and Doyle,[30] Heuss,[34] Krüger,[40] Swynghedauw and Gaudier[72]).

CASE REPORT.

HISTORY.—A man, aged 45 years, ten days previous to his admission to the hospital, while felling trees had been struck a glancing blow in the left upper abdominal quadrant by a branch of a falling tree. He insisted that the blow was of moderate severity, that he was not knocked down, and that no external injury resulted. Almost immediately, however, he experienced violent pain in the left upper quadrant which lasted about ten minutes. He was then able to continue work although slight pain persisted for the remainder of the day. In the night it again became severe, and the next day he consulted his physician. By that time the pain had practically disappeared and nothing of note was found on examination. Accordingly he worked for seven days, although he still complained of continuous nagging pain in the injured area. On the morning of the ninth day after the original injury he was suddenly seized with a violent exacerbation of the same pain, this time extending through to the back and up to the left shoulder. He became nauseated but did not vomit. He had considerable pain on deep inspiration at the base of the left lung. Within a short time he collapsed. In the previous nine days he had not noticed fever or any urinary abnormality. The previous history was entirely negative.

ON ADMISSION.—When admitted to the clinic the man was gravely ill; his skin was pale and covered with cold sweat, his eyes were lustreless, and his attention wandering. His respiration was rapid, grunting, and purely costal; his knees were flexed on his abdomen for greater comfort. The pulse-rate was 100 and the temperature 99·2°; the blood-pressures were 140 and 80 (normal blood-pressure 200 systolic). The diaphragm was elevated on both sides, particularly on the left, with fluid at the left base and moderate bronchopneumonia on both sides. There was suspicious tympany in the left side of the thorax, which rather suggested the possibility of a diaphragmatic hernia.

The abdomen was generally rigid and tender, especially in the left upper quadrant, where an indefinite mass could be felt, which was dull to percussion, from the seventh costal cartilage to the level of the umbilicus. The dullness did not shift. Rectal examination was negative. The urine was normal. The hæmoglobin was 48 per cent; erythrocytes numbered 2,720,000 and leucocytes 25,800; the differential count was negative. A diagnosis was made of concealed abdominal

hæmorrhage probably from ruptured spleen and possibly left-sided diaphragmatic hernia. The patient was observed for twelve hours, and the severe dehydration was combated by solutions of saline administered subcutaneously. The next day it was obvious that the patient was worse and operation was performed.

OPERATION.—Under local and ethylene anæsthesia the abdomen was opened by a wide transverse incision in the left upper quadrant. Blood was immediately encountered inside the peritoneum. Further exploration revealed a huge hæmatoma, consisting of old black clots and recent hæmorrhage, extending over the stomach from the transverse colon below to the left dome of the diaphragm above. It probably contained about 2 litres of blood. A piece of spleen was next encountered about 8 cm. in diameter, attached by a fragment of capsule to what was taken to be the remains of the pedicle of the spleen. There were no blood-vessels, however, to be noted in this fragment of capsule. The remainder of the spleen, in five separate pieces, was found loose in the blood-clot and held together by it. The splenic pedicle appeared to be entirely free, but active hæmorrhage was not occurring from it. The whole mass was quickly removed from the abdomen and three clamps were placed across the splenic pedicle as a protective measure. A few bleeding veins on the greater curvature of the stomach were ligated. The cavity was packed with iodoform gauze and the abdomen was closed. The patient was given a transfusion on the table of 500 c.c. of citrated blood, and immediately was placed in an oxygen tent, where he remained forty-eight hours. The clamps were removed at the end of this time and the packing was removed in seven days. Convalescence was uneventful, and the patient was up and about on the sixteenth day after operation.

PATHOLOGY.—Section of the largest of the five fragments of the spleen showed such extensive subcapsular hæmorrhage that almost the entire capsule had been

FIG. 99.—Spleen in five fragments as removed from the perisplenic hæmatoma. Note the extensive subscapular extravasation of blood. The lighter area of the largest piece represents normal spleen.

stripped up and separated from the subjacent parenchyma by uniform firm layers of partially organized blood-clot from 0·5 to 1 cm. thick ; this process was observed in each fragment of spleen (*Fig.* 99). There also appeared to be some deeper parenchymatous hæmorrhage with evidence of thrombosis and early scattered infarcts. In other places perfectly normal splenic pulp could be seen. The ruptured surfaces seemed to be of more recent origin, owing to the lack of organizing blood-clot.

Microscopic examination of the intrasplenic blood showed definite attempts at organization with numerous fibroblasts and endothelial loops sprouting from the normal splenic pulp to invade the extravasated blood-clot (*Figs.* 100, 101).   The amount of organization which had occurred was compatible with a lesion of eight

FIG. 100.—The edge of the intrasplenic hæmatoma.   Active organization is proceeding.   Endothelial buds and fibroblasts can be seen invading the blood mass from the adjacent splenic tissue.   (× 110.)
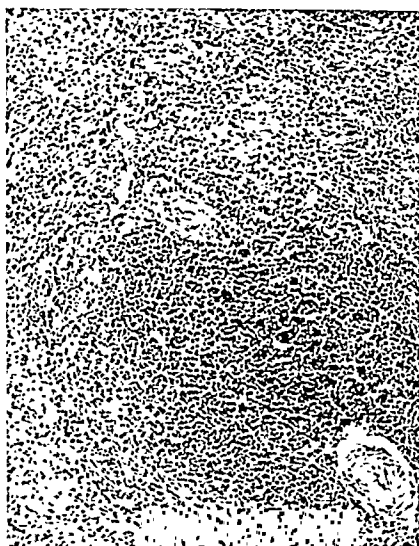
FIG. 101.—Normal spleen in a section taken from the centre of the fragments. (× 110.)

FIG. 102.—Subcapsular hæmatoma.   Some splenic tissue is left attached to the capsule during the stripping process.   The capsule is in reality forcibly torn from its attachment and not stripped.   (× 100.)

to ten days' duration. On the other hand, the blood from the perisplenic hæmatoma was of much more recent origin : organization was not observed here. A small layer of normal splenic pulp was attached to the capsule torn away by the hæmorrhagic extravasation (*Fig.* 102). It appeared clear that this injury was an example of the formation of a primary intracapsular hæmatoma with extensive subcapsular hæmorrhage followed by attempts at healing over a period of eight days. Repeated slow hæmorrhages, however, had apparently increased the intracapsular tension to the point at which a slight rise in intra-abdominal or intravascular pressure had caused secondary capsular rupture of explosive violence which shattered the spleen entirely and blew it off its pedicle. The huge perisplenic hæmatoma was then formed. No doubt, the profuse bleeding which followed the secondary rupture aided materially in further disintegrating the organ.

CLINICAL FEATURES.

Age and Sex.—The spleens of males are more likely to be injured than those of females, since in the active period of life males are more subject to blows, bumps, falls, and kicks. The youngest patient on record was aged 8 years, and the oldest 63. Most of them were between the ages of 15 and 50. Only 4 of the 46 patients were women. Schlegel observed that if free blood is found on opening the peritoneum of a male patient, rupture of the spleen should first be considered. This takes into consideration the fact that spontaneous rupture of the abnormal spleen is by no means uncommon. Ruptured ectopic gestation should be considered in women.

Initial Injury. — It is well known that a patient who presents the usual signs of ruptured spleen has almost always suffered an accident of great severity, although notable exceptions to this rule occur. The reasons for this may be sought in the anatomical position of the viscus. The spleen is freely movable on its pedicle ; it is protected by the elasticity and cushioning effect of the neighbouring viscera and lies hidden beneath the ribs so that it is adapted to resist external violence and to retreat before shocks and blows. The evidence for the severity of the violence required to produce rupture of its capsule is to be found in the frequency with which neighbouring structures are coincidentally involved. One may, therefore, surmise that the force which produces uncomplicated rupture of the normal spleen is usually sudden, severe, and localized to the splenic region. In the so-called subcutaneous injuries, besides the effect of the direct blow, the organ is hurled suddenly against the vertebral column and crushed or ruptured by contrecoup violence. This explains the frequency of involvement of the hilum and consequent profuse hæmorrhage. If the spleen is pathologically enlarged it no longer lies protected, but projects forward directly beneath the anterior abdominal wall, where it is exposed to blows. The friability of the pulp, its extreme vascularity, the brittle nature of the capsule from hyaline perisplenitis, and the frequent fixation of the organ to the parietal walls make it exceedingly vulnerable to external trauma. Under such circumstances the slightest increase in intra-abdominal pressure, such as that caused by coughing, vomiting, bending, or straining at stool, may be sufficient to burst a splenic vessel. So-called spontaneous rupture of the spleen, a common abdominal catastrophe in malarial regions, is explained in this way. The same accident has been reported in leukæmia,[33] ulcerative endocarditis,[38] typhoid and typhus fever, influenza,[26]

and carbuncle.[22] Wild reported the cure of a hæmorrhagic diathesis (apparently essential thrombocytopenic purpura) following splenectomy for spontaneous rupture of the spleen. In connection with such spontaneous ruptures Camus quoted the aphorism of Besnier to the effect that "traumatism plays a part in spontaneous rupture of the spleen and the spleen is ready to rupture when the trauma occurs."

The initial injury to the spleen in this condition, however, is of necessity comparatively slight, for temporary recovery from it always occurs within a short time. In 44 cases in which the primary injury was recorded, 20 accidents were severe, 16 were moderately severe, and 7 were slight ; in 1 case rupture apparently was spontaneous. In all but one, the force of the blow was directed against the base of the left side of the thorax or the left upper abdominal quadrant. The single example of indirect injury was furnished by Hahn's case, in which the patient was thrown violently to the ground, striking the right side of the abdomen.

The symptoms produced by such accidents varied with the severity of the injury. The preponderance of the complication of fractured ribs over all others (10 cases), especially those of the lower left side, should always arouse suspicion of subjacent splenic injury. More often than not, the associated injuries completely masked the underlying condition. As almost invariably happens when the abdomen receives a severe blow, the patient immediately suffered from a varying degree of traumatic shock. Dizziness, faintness, weakness, nausea, and vomiting, or even complete collapse associated with rapid pulse, pallor, clammy skin, cold extremities, and shallow respiration, were noted. Recovery from this in favourable cases occurred in twelve or fifteen minutes, and the patient could then be helped to his home, or he walked to the nearest hospital. Sometimes he was even able to go about his affairs. If the injury was slight or only moderately severe, these general symptoms were usually entirely absent, so that within a few days all recollection of the accident had passed from the patient's mind. In this connection one might speculate on whether the seven remarkable cases of spontaneous rupture of the normal spleen reported by Susman and others were not in reality examples of secondary hæmorrhage following accidents which had entirely escaped the patient's notice.

The closest attention, however, should be paid to the local symptoms and signs, for these furnish the only definite evidence of splenic injury. Pain over the region of the spleen was a complaint in almost every case. It varied in intensity from mere discomfort or dull aching to severe stabbing paroxysms of great intensity forcing the patient to lie down until relief was obtained. Once only did the pain radiate to the left shoulder. Possibly careful questioning would have elicited this symptom more often, for its significance in lesions in the vicinity of the diaphragm cannot be exaggerated. Abdominal rigidity, generalized at first, but more often localized to the epigastrium or to the left upper abdominal quadrant, was the commonest sign, and was associated with a varying degree of tenderness both here, in the left costovertebral angle, and at the base of the left side of the thorax (*Fig.* 103, 1). Rigidity in abdominal contusions not limited to the injured point is of paramount diagnostic significance and is a clear indication for immediate laparotomy. Suspicion of

underlying injury to the spleen should therefore always be entertained in spite of the presence of other injuries if after such an accident persistent abdominal rigidity is found in the left upper abdominal quadrant. In a certain number of cases in this series symptoms or signs were not present, beyond a little discomfort or dull ache in the left side, but the spleen had undoubtedly ruptured. Diagnosis in such cases was manifestly impossible. The patients would seem to be in great danger from the almost inevitable
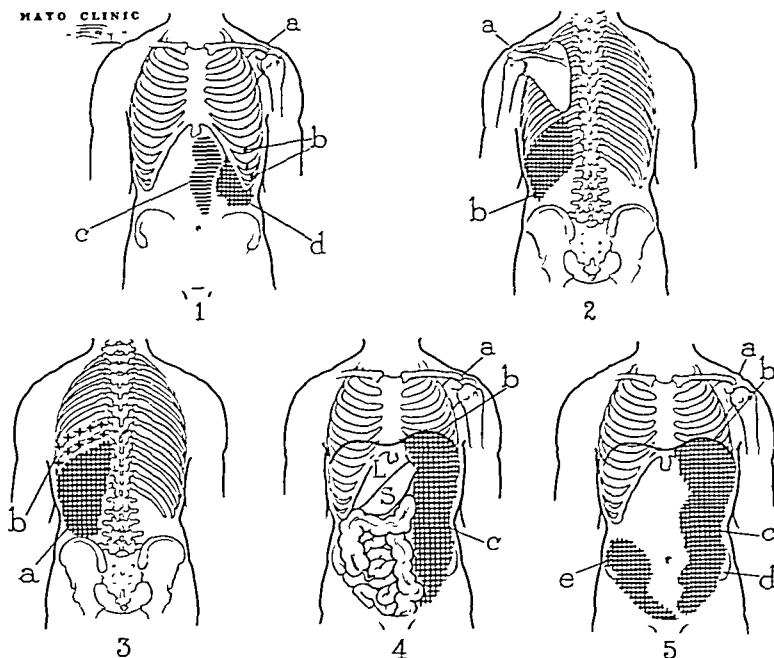
Fig. 103.—1, The initial injury; no increase in splenic dullness: (a) Reflex pain in the left shoulder; (b) fractured ribs; (c) left upper abdominal rigidity; (d) pain and tenderness. 2, The initial injury; slight increase in splenic dullness posteriorly; probably capsular rupture with perisplenic hæmatoma: (a) Reflex pain in shoulder; (b) increased splenic dullness. 3, Secondary rupture: (a) Large perisplenic hæmatoma from posterior aspect; (b) occasional involvement of left lung and pleura through diaphragm. 4, Secondary rupture; large perisplenic hæmatoma from anterior aspect showing displacement of organs: (a) Pain in shoulder; (b) elevation of diaphragm; (c) perisplenic hæmatoma. 5, Secondary rupture; large perisplenic hæmatoma spilling over into peritoneal cavity: (a) Reflex pain in left shoulder; (b) elevation of diaphragm; (c) perisplenic hæmatoma; (d) fixed dullness; (e) shifting dullness.

secondary rupture, for when it occurs they are likely to be far from medical aid.

The duration of the disability occasioned by the initial injury depended more on the associated injury than on splenic trauma and the cessation of subcapsular or perisplenic bleeding. Many subjects returned to work in one or two days, while a few complained of acute symptoms referable to the traumatized spleen for only a few hours. Most patients did not return to work entirely free from symptoms, but they were able to work.

Latent Period.—In the group of cases in my review the latent period began with the cessation of splenic bleeding and was terminated by the abrupt onset of secondary bleeding at a date remote from the primary injury. In the usual case of rupture of the spleen the swift appearance of the signs of internal bleeding does not coincide with the onset of a fresh, fulminating hæmorrhage but represents either the rapid failure of the patient's resistance against an internal hæmorrhage, profuse and continuous from its onset, or collapse, following the sudden evacuation of a constantly augmented perisplenic hæmatoma into the general peritoneal cavity. This rather short intervening period is to be distinguished from the true latent period in that here all bleeding has ceased and the interval of comparative symptomatic silence is prolonged. The shortest period was forty-eight hours, the longest six months. As may be noted in *Table I*, hæmorrhage occurred most often between the third and ninth days, rarely after the fourteenth day.

Table I.—Frequency of Secondary Rupture according to Latent Period.

LATENT PERIOD	CASES
2–6 days ..	23
7–11 ,, ..	12
12–16 ,, ..	4
17–21 ,, ..	3
22–26 ,, ..	1
26–30 ,, ..	1
More than 30 ,, ..	1

During the latent period an excellent opportunity may be afforded of confirming a suspicion that the spleen is ruptured, of guarding the patient against secondary hæmorrhage, or even of operating before it occurs. Many authors have observed that although complete freedom from all symptoms was occasionally undeniable, in certain cases symptoms did exist but escaped notice because the patient did not complain of them. Even though patients were attending to their daily affairs, absolute symptomatic silence was far from the rule. The two elements of most significance were persistence of a dull remittent or exacerbating pain in the left side and of slight rigidity in the left upper abdominal quadrant (*Fig.* 103, 1). The latter was provoked by the slightest examination and was occasionally present when the pain was minimal. Repeated examination was often necessary to be sure of its presence. It was Perrin's belief that if the rigidity increased, a more serious lesion than simple contusion should be suspected. Less frequently slight acceleration of the pulse-rate was demonstrable (80 to 100), but this was noted only if a perisplenic hæmatoma had formed at the primary rupture.

Costal breathing was mentioned by several writers, and, as in Strauss's case and in mine, signs of left basal pleurisy or pneumonia were suggestive. Fever was distinctly uncommon, but the leucocytosis of hæmorrhage was not infrequent. In a few instances more definite signs indicated the existence of a perisplenic hæmatoma. such as fullness in the left hypochondrium and an increase in splenic dullness associated with slight pallor. weakness, dizziness. and occasional fainting spells (*Fig.* 103, 2). These, however, were more characteristic of the cases in which bleeding was slowly progressive, the patient gradually going from bad to worse. Percussion is of relatively little value in determining the size of the spleen. Normally there is dullness in the mid-axillary line from the ninth to eleventh ribs corresponding to that part of the organ which is most superficial. Its anterior edge of dullness is approximately in the anterior axillary line. If this is enlarged upward and forward beneath the anterior abdominal wall. or downward posteriorly below the eleventh and twelfth ribs, it is probable that a small perisplenic hæmatoma is present.

Secondary Splenic Rupture and Delayed Hæmorrhage.—The abrupt onset of delayed hæmorrhage immediately constituted in all cases a grave abdominal disaster, made more serious by the apparent lack of an adequate cause, by the difficulty in diagnosis. and by the fulminating character of the bleeding. The time of onset—forty-eight hours to six months after the initial injury— apparently had nothing to do with the physiological state of contraction or expansion of the spleen. In several cases it occurred while the patients were going quietly about their affairs without effort. On the other hand, it took place several times just after slight strain, such as that attending defæcation, pulling on socks, and so forth. Very little was required to break down whatever protective barrier might have formed around or within the injured organ ; sometimes a slight effort produced it, but just as often it was apparently spontaneous. It took place as soon as the intrasplenic vascular pressure was too great for the weakened splenic tissue and perisplenic adhesions to sustain it.

The onset of hæmorrhage was almost always followed by the same characteristic train of symptoms. A sudden agonizingly severe attack of pain was felt, usually in the left side or more diffusely in the abdomen, and associated with abdominal rigidity, tenderness, and a varying degree of shock even to complete loss of consciousness, pallid sweating skin, cold extremities, rapid feeble pulse, and unobtainable blood-pressure. Death occurred with startling rapidity in seven cases before anything could be done. Such an event taking place just after the patient had been allowed out of bed was suggestive of pulmonary or mesenteric embolism or coronary thrombosis.

If the patient survived the initial collapse, the diagnosis of severe internal hæmorrhage was not long in doubt. unless the short subsequent period of improvement already mentioned made added difficulties. Increasing pallor and weakness, rising pulse and falling blood-pressure, dyspnœa and costal breathing, with nausea and vomiting, were almost constantly found. If the blood was examined in the laboratory, a marked drop in hæmoglobin and in the number of erythrocytes, combined usually with a sharp rise in leucocytes, occurred. The simultaneous occurrence of abdominal rigidity and tenderness,

with increasing dullness in the left side or in both flanks, usually made it clear that serious intra-abdominal hæmorrhage was occurring.   Distinct fullness in the left hypochondrium sometimes amounting to a definite mass was occasionally noted.   In my own case W. J. Mayo called my attention to the sensation of doughiness or soft lumpiness imparted to the examining hand from the presence of firm clots in the hæmatoma.   Such symptoms were of course entirely sufficient to justify immediate abdominal exploration, but they offered little help as to the source of the hæmorrhage, a point of considerable significance in placing the exploratory incision.   Pitts and Ballance stated that a splenic origin could be deduced " from the locality of the injury, from the evidence of internal hæmorrhage, from the great increase in fixed dullness in the splenic region, and from the fact that though both flanks were dull on percussion the right flank alone became entirely resonant on change of position."

The value of 'Ballance's sign', as it is now called, has been questioned by several observers because typical signs of free fluid were repeatedly obtained in this condition.   It seems obvious that the sign could be elicited most easily during the period of expansion of the splenic pouch by blood, and while the growing perisplenic hæmatoma was localized.   At some time during the evolution of this clinical picture it should be found.

Pain in the left shoulder referred from the splenic region was noted in several cases.   Its occurrence is probably more frequent than is generally believed, owing to the ease with which it is overlooked.   Another observation of considerable assistance in the diagnosis was elevation of the left diaphragm from the pressure of the subjacent hæmatoma.   Naturally in such emergency little time is given for the taking of skiagrams of the thorax.   So far as I know, skiagrams were not made in any case except mine (*Fig.* 103, 3, 4, 5).

Atypical symptoms were occasionally noted.   Thus in Perrin's second case the perforation of a hollow viscus seemed responsible.   The same diagnosis was made in Stolze's case.   An even more marked example of this occurred in Eisenklam's case, in which the abdominal pain, tenderness, and rigidity were all on the right side.   In Wohlgemuth's case the patient appeared to have suffered rupture of a Fallopian tube, because the pain was low on the left side and the initial injury to the spleen was not recognized before operation.

In general one might state that the diagnosis may be made from a knowledge of the injury and the site of the primary injury ; from the presence of a persistent dull ache in the left side with slight left upper abdominal rigidity during the latent period ; from the later sudden onset of signs of severe internal hæmorrhage, and in conjunction with the appearance of Ballance's sign, elevation of the left diaphragmatic dome, or pain in the left shoulder referred from the splenic region.

PATHOLOGICAL FEATURES.

Initial Injury.—Almost an exact parallel may be drawn between this condition and that of ectopic pregnancy, in which delayed hæmorrhage is notoriously common.   Except for the fact that trauma produces in the spleen

what occurs spontaneously in the tube, the pathological evolution of the perisplenic hæmatoma and the pelvic hæmatocele is almost identical. Both, however, for anatomical reasons differ greatly from retroperitoneal hæmatoma resulting from such injuries as ruptured kidney. In contradistinction to the so-called spontaneous rupture of the spleen, predominantly an affection of the pathological organ, this accident occurred almost exclusively in the normal spleen (*see Fig.* 102). Of the 46 cases, malaria was present in only 2. The primary injury to the spleen may be classified in three groups : (1) Minor superficial capsular rupture or slight splenic contusion producing parenchymal ecchymosis ; (2) Intrasplenic hæmatoma and subcapsular hæmorrhage without capsular rupture ; and (3) Capsular and parenchymal rupture with perisplenic hæmatoma.

Minor superficial ruptures of the capsule or slight contusions of the spleen are probably not so rare as is generally believed, but they are undoubtedly far from common. They are merely small fissures in the capsule with little, if any, involvement of the subjacent parenchyma, and although they may be the cause of abundant hæmorrhage, usually they are not. Indeed, bleeding may be slight. Collin has called such lesions 'dry ruptures'. This is particularly the case if the lesion is situated on the convex surface of the spleen. What determines the severity of the hæmorrhage is not the depth of the rupture but the importance of the vessels sectioned. Thus fissures in the neighbourhood of the hilum are much more likely to produce severe hæmorrhage than in any other situation, and furthermore they show the least tendency to localization.

Splenic contusions consisting of small ecchymoses in the subcapsular region but without any extensive disorganization of the parenchyma or localized formation of hæmatoma are also in the main of minor significance. They undoubtedly heal rapidly, leaving scarcely any appreciable trace of their existence beyond a little scarring of the capsule or localized perisplenitis with adhesions.

Splenic contusions and superficial tears, unless they involve the hilum, tend toward spontaneous cure by simple scar formation. Although it is difficult to determine how many secondary hæmorrhages followed such superficial lesions a careful consideration of the cases in this series leads me to the conclusion that few, if any, occurred. Most of the primary injuries were of a somewhat more serious nature.

In most cases of intrasplenic hæmatoma and subcapsular hæmorrhage without rupture of the capsule, if the spleen was studied after operation or at necropsy, it was noted that diffuse or localized subcapsular hæmorrhage or more deeply placed hæmatoma formation was perhaps the most striking feature of the condition. If it was well marked where the capsule remained intact it was less obvious where intrasplenic tension had been relieved by fracture of the capsule at the primary injury. In about half of the cases microscopic examination showed that intrasplenic bleeding occurred before the initial rupture of the capsule, and was therefore to be regarded as the primary lesion. In the remainder of the cases some degree of concomitant rupture of the capsule had occurred, and the intrasplenic hæmatoma, if present, was associated with a larger or smaller perisplenic hæmatoma, the

evolution of which will be considered later. If the capsule remained intact, however, the trauma apparently produced true subcapsular or parenchymal rupture, giving place to a hæmorrhage whose tendency to assume large proportions was limited only by the tensile strength of the capsule. The resulting extravasation of blood into the place where the spleen pulp was broken contained in addition an admixture of pulp débris lying in a network of partially ruptured and stretched trabeculæ. The walls were ragged and irregular and were infiltrated with blood for a considerable distance from the hæmatoma itself.

In six cases (Ganguli,[31] Lejars,[41] Lempriere,[42] Pitts and Ballance,[59] Schlegel,[64] Swynghedauw and Gaudier[72]) the hæmatoma took the simplest form of a fairly well localized cystic mass, the so-called blood cyst, and produced a distinct purplish bump on the surface of the organ. These cysts
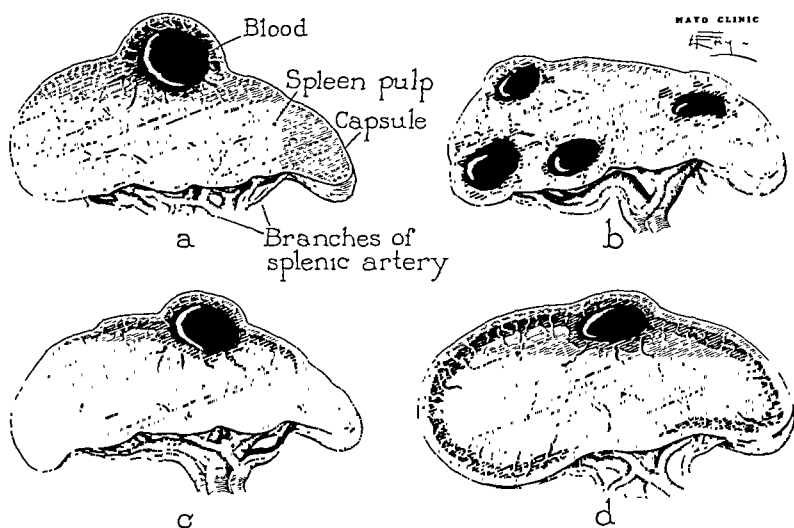
FIG. 104.—a, Intrasplenic hæmatoma—solitary 'blood cyst'; b, Multiple intrasplenic hæmatomata; c, Intrasplenic hæmatoma with moderate subcapsular hæmorrhagic extravasation; d, Intrasplenic hæmatoma with complete subcapsular extravasation.

varied in size from 2 to 8 cm. in diameter, and except for the case of Swynghedauw and Gaudier were single (*Fig.* 104, *a*, *b*). They were not situated in any particular part of the spleen, but seemed to acquire added hazards in the region of the vessels of the hilum.[31] In Lejars' case the whole spleen had been converted into a blood cyst by the complete disorganization of the parenchyma, and eventually it attained enormous proportions. In fifteen cases the original injury consisted of the same localized intrasplenic collection of blood, but here either by reason of its progressive burrowing capacity or because the subcapsular splenic pulp was particularly likely to be subjected to a tearing or crushing injury, the extravasated blood showed a marked tendency to spread laterally beneath the capsule and to lift it away from the subjacent parenchyma (*Fig.* 104, *c*). This was not true stripping of the capsule, but rather a tearing of the firm union which normally exists

between the fibrous splenic capsule and the trabeculæ supporting the pulp tissue. Some splenic tissue was thus left attached to the elevated capsule (*see Fig.* 102). The lateral extent of this hæmorrhagic extravasation varied from a few centimetres to complete separation of the capsule from the underlying pulp as far as the vessels of the hilum[15, 67] (*Fig.* 104, *d*). Why this area is so prone to hæmorrhagic extravasations is not known, but it is possibly related to the fact that this is the area of greatest movement of physiological volume. Lubarsch and Wolff assumed that this subcapsular area represents a 'flood region' of displacement of blood, and that injury is more likely to affect the organ in this region than elsewhere. The practical effect of the process was to increase enormously the area of the tensely stretched capsule, whose impaired nutrition would predispose toward secondary rupture. Whether the result of the primary injury was a single localized hæmatoma, multiple hæmorrhagic foci, or these two combined with extensive subcapsular extravasation, the cessation of bleeding depended entirely on the strength of the capsule. Splenic deformity and enlargement slowly progressed until the pressure exerted by the distended capsule equalized that produced by the extravasating blood. With the cessation of intrasplenic bleeding the patient entered the latent phase of the condition.

As has been pointed out, there was about an equal number of cases in which the predominating lesion was a frank capsular and parenchymal rupture with perisplenic hæmatoma. I am not considering here those cases in which the capsule alone was injured, but those in which more extensive involvement occurred. In many of these subcapsular bleeding developed at the same time, but since it was rarely so extensive as when the capsule remained intact, and as the secondary hæmorrhage was usually found to have occurred at the site of the already present rupture, this factor was not of great significance. The combined capsular and parenchymal ruptures were of every conceivable shape, size, and depth, and were not confined to any particular part of the organ, except so far as the concave surface was injured more frequently. Naturally the convex surface and posterior border were most commonly involved. The hilum as a site for secondary rupture was rather unusual, probably because any injury here involves important vessels which bleed furiously and show little tendency to localization. In many cases the lesions were multiple, both surfaces being affected. They varied from straight, curved, or angular to star-shaped, and extended into the parenchyma from a few millimetres to the entire thickness of the organ.

The rupture generally contained between its two surfaces a particularly fibrinous blood-clot attached to larger bloody masses which formed the perisplenic hæmatoma. Some amount of early cicatricial organization was usually apparent at the margins of the wound, the edges being somewhat indurated, covered with plastic lymph, and loosely adherent to surrounding organs, to omental tags, or to blood-clots. It was thus possible to estimate roughly the age of the rupture, particularly in relationship to any concomitant lesions as well as to the site of secondary hæmorrhage. The pathologic course of these primary rents in the spleen from the time of their infliction to the temporary cessation of bleeding depended on a number of variable factors. The more positive hæmostatic value of the capsule being here lost, other means

of controlling and localizing the resultant hæmatocele were utilized. To allow time for these less dependable agencies to swing into action, it is essential that the primary hæmorrhage be slow.

In many instances reference was made to the tamponading effect of clots between the two walls of the rupture (Buxton,[13] Jackson,[36] Kroner,[39] Neck,[51] Noetzel,[52] Wohlgemuth[77]). It was further noted, however, that in such a vascular organ as the spleen the weak fibrinous union which they produced between two such soft and friable surfaces offered little hope of efficient hæmostasis. With the spleen in his hand Perrin was able to observe how easily they might be dislodged and how easily bleeding might be recommenced. Old and recent perisplenic adhesions had probable value in limiting the expansion of the splenic pouch and in bringing such organs as the diaphragm, stomach, colon, or omentum into close apposition with the wound. Few, if any, adhesions could have existed before the primary rupture because in only two cases was there evidence of pre-existing pathologic processes. The
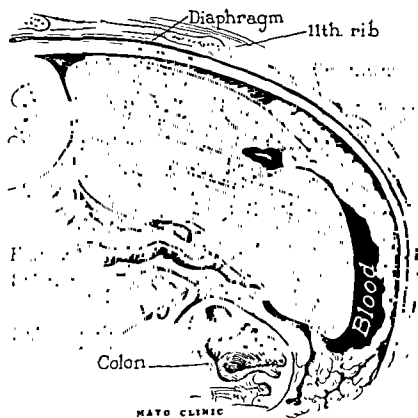
FIG. 105.—Capsular and parenchymal rupture with small perisplenic hæmatoma. Rent plugged with blood-clot and omental tongue.

frequency with which a tongue of omentum had insinuated itself between the margins of the rupture or had plastered itself over the wound was striking. The omentum itself was usually found to be blackened, swollen, and infiltrated with old blood. The early union was rather fragile,[57] but later was firmer and less easily separated. Omental plugging combined with the tamponading effect of the subjacent clots offers the most efficacious and most usual method of immediate and temporary hæmostasis (Fig. 105).

In addition to these hæmostatic agencies it is necessary to examine the anatomic disposition permitting the formation of a perisplenic hæmatoma, which either remains local or finally ruptures into the general peritoneal cavity. Constantinesco explained why a perisplenic hæmatoma may attain tremendous proportions before peritoneal inundation occurs. The spleen, like the Fallopian tubes but unlike the kidneys, is surrounded by a potential space bounded on all sides by organs or structures easily displaced so that a cavity capable of containing the entire volume of blood can be formed.

When the extravasation of blood took place at the primary rupture it rapidly surrounded the spleen, elevated the diaphragm above, from which respiratory symptoms occasionally arose, displaced the splenic flexure of the colon toward the iliac fossa, the stomach and intestinal coils to the right, the parietal wall of the left hypochondrium laterally and behind, and the omentum and the anterior abdominal wall in front. The presence of the gastrosplenic and gastrocolic ligaments and the mesocolon further aided in preventing the diffusion of blood to the right and above, whereas, as Trendelenburg has pointed out, reflex contraction of the anterior abdominal wall on the subjacent omentum and coils of small intestine greatly contributed to the localization of the hæmatoma anteriorly. Provided the hæmorrhage was reasonably slow, adhesions soon formed between the omentum and stomach, and the omentum and anterior abdominal wall, while the coils of small intestine and the omental tags of the colon stuck together in the vicinity of the hæmatoma. Thus the defensive reaction of the peritoneum by coalescence of the various organs which appeared more or less infiltrated with blood, produced an enclosure which did not permit diffusion of hæmorrhage and peritoneal flooding and aided materially in limiting further bleeding. Nevertheless, a true cyst was not formed and, with the exception of Lejars' case, trace of a capsule could not be found. These fibrinous adhesions were easily broken down. In few, if any, of the cases of primary hæmatoma was the tumour of large size, and in six only (Cohn,[18] Freund-Kirchner and Doyle,[30] Ganguli,[31] Kroner,[39] Noetzel,[52] Strauss[69]) was its presence manifested by clinical signs such as increased splenic dullness, Ballance's sign, or free fluid in the abdomen. The extravasated blood, at first fluid and of dark colour, rapidly coagulated to form a mass of black clots bathed in a considerable quantity of serum. Much of the latter undoubtedly seeped down through the peripheral adhesions to reach the pelvis and lower part of the abdomen and produced signs of free abdominal fluid. The liquid part of the hæmatoma soon became thick, syrupy, and brownish-red. Its resemblance to treacle was frequently noted.

Latent Period.—The time taken for hæmorrhage to cease probably varied largely with the type of injury. Thus hæmostasis would be more or less complete in intrasplenic lesions within a few minutes or hours. If extensive subcapsular extravasation was occurring, the process would be prolonged perhaps over a period of days, but at a much slower rate and probably intermittently after the initial immediate bleeding had been checked by the resistance of the capsule. Following capsular rupture and perisplenic bleeding, hæmostasis was more difficult to establish and maintain. It is obvious from the history of frequent attacks of left upper abdominal pain and tenderness with progressive weakness, anæmia, and slight increase in splenic dullness during the latent period, that sometimes fresh bleeding occurred intermittently and that the primary hæmatoma gradually increased in size. In most cases, however, hæmostasis was temporarily complete and the period of latency was occupied by attempts at healing. This phase of the question has been studied by Bland-Sutton, Englmann and Hitzler, and others. My case showed almost every stage of organization and repair, interrupted, it is true, by secondary rupture. Healing occurred by scar formation in the ordinary way. Fibroblastic proliferation of the splenic capsule, the trabeculæ, and

the adherent omentum sealed off the rent in the capsule concomitantly with the endothelial and fibroblastic invasion of the included blood-clot, thus replacing the earlier fibrinous adhesions by means of which primary hæmostasis was effected. The reticular and lymphoid elements of the parenchyma also showed marked proliferative activity. The same process was to be noted in the adhesions surrounding the perisplenic hæmatoma, these becoming consolidated by fibrous tissue infiltration. Struckhof observed the healing process in the spleen experimentally and has found it to be identical with what I have described.

In such a soft vascular organ as the spleen, and situated in such a locality, the balance maintained between the *vis a tergo* of the extravasating blood and the hæmostatic forces opposed to it was an exceedingly delicate one, for in all cases a little increase in the former was sufficient to bring about secondary rupture. The amount of trauma required has already been considered.

Besides the complication of delayed hæmorrhage, four varieties of termination of the healing process are recognized both in intrasplenic and perisplenic hæmatoma.

1. Complete absorption of the blood-clot and healing of the solution of continuity by scar tissue is more likely to occur if the initial injury is slight.

2. Apart from secondary rupture, encystment is undoubtedly the most common termination, especially if any subcapsular extravasation has occurred. The encysted hæmatoma or traumatic blood cyst acquires a fibrous-tissue capsule devoid of endothelial lining, whereas the contents gradually become serous, serosanguineous, or remain definitely hæmorrhagic. In practically every case blood pigment is a prominent feature of the walls of the cavity.

3. In certain rare cases, and then only in intrasplenic hæmatoma, the serum is absorbed, while the solid portion of the clot persists and becomes progressively harder. The primary hæmatoma is thus transformed into a firm mass of greyish-white tissue described under the name of ' fibrinous tumour of the spleen.' [14, 60]

4. Suppuration in the hæmatoma, also a rare complication, has occasionally been described.

Secondary Hæmorrhage. — The mechanism by which this complication was brought about varied with the original injury. In the case of an intrasplenic hæmatoma the tensely stretched capsule, weakened by the force of the original blow and softened by the inflammatory reaction following the trauma, finally gave way at its most vulnerable point. The free escape of blood into the splenic pouch and general peritoneal cavity was thus permitted. If temporary hæmostasis had been effected in a frank splenic rupture, a little effort on the part of the patient was sufficient to start the bleeding again. In either case, bleeding, once restarted, was profuse and uncontrollable. In seven cases it was so overwhelming that death occurred within a few minutes ; the peritoneal cavity was literally flooded with blood. In most cases, however, rapid and tremendous expansion of the splenic pouch took place, the hæmatoma eventually occupying the entire left side of the abdomen and extending down into the pelvis. Not infrequently this enormous collection

of blood remained localized to the end, none escaping into the general peritoneal cavity. In none except Lejars' case was a true cystic condition found. Well-marked localization was particularly the case if secondary hæmorrhage had taken place in an already existent perisplenic hæmatoma. Even here blood began to spill over as a terminal event. More often the severity of the hæmorrhage caused general peritoneal involvement coincidently with the filling of the splenic pouch, and at operation or necropsy massive clots surrounded the spleen and were distributed throughout the intestinal coils and abdominal fossæ. Patel and Vergnory felt that most of the severe abdominal symptoms arose from the moment general peritoneal extravasation began rather than from bleeding into the splenic pouch. The topographical features of the perisplenic hæmatoma have been considered.

An interesting point with regard to the spleen itself remains to be dealt with. In the case I reported the spleen was found at operation to be in five distinct fragments lying free in the hæmatoma (*see Fig.* 99). Burger found the spleen in three separate pieces. In the cases of McCaw, Pétridis, Wohlgemuth, Patel and Vergnory, and Nast-Kolb, the spleen was practically divided into halves. Most of the other reports recorded the fact that the ruptures found at operation or necropsy were obviously much larger and more extensive than those which could have been inflicted at the original injury. It seems clear that secondary hæmorrhage occurred with considerable violence and that it had a marked shattering and disintegrative effect on the spleen, sufficient even to bring about complete separation of the organ from its pedicle. Whether this occurred immediately after the onset of secondary bleeding, or whether it was a more gradual process, is difficult to determine. The finding of such an extensive lesion at operation without an adequate cause should not blind one to the fact that it had its genesis in a much smaller injury and at a date previous to that of its most obvious clinical manifestations. One should not make the mistake of believing this to be a spontaneous splenic rupture, but rather the end-result of a forgotten or disregarded injury.

TREATMENT.

The operation of choice for rupture of the spleen is splenectomy. Any form of repair or tamponment is inadmissable. Of the thirty-seven cases in which operation was performed, splenectomy was done in every instance, with the exception of Lejars' case, in which drainage of the encysted hæmatoma was carried out. Apart from the almost certain fatal outcome without surgical intervention, the poor results of medical treatment are illustrated by the case reported by von Kolb. Here, despite repeated urging, the patient absolutely refused operation. Convalescence, remarkably achieved, was slow and stormy, and after seven months, with only partial absorption of the hæmatoma, the patient was unable to work, and even walking about required several hours of rest every day.

Since thorough exposure is necessary, splenectomy is best performed through the wide transverse left subcostal incision described by Ruggi and practised by Pauchet and C. H. Mayo. The left upper rectus approach, described by Bevan (Bevan's incision) and Balfour, is also satisfactory.

Transfusion, gum acacia, and oxygen post-operatively are of the greatest benefit and may even be life-saving. Whether one should perform splenectomy before secondary rupture has occurred and during the latent period is a matter for careful consideration. Perrin, who had the happy experience of successfully anticipating a secondary rupture, was strongly of the opinion that it should be done if possible. From a study of the risks involved I am inclined to agree with him. Special emphasis, however, should be laid on the significant point that a patient who has sustained an injury of the left side, particularly with fracture of the ribs, and if there is a suspicion of rupture of the spleen, should be kept at rest and under close observation for at least fourteen days. Sudden and apparently spontaneous internal hæmorrhage could then be attended to with dispatch and with a more certain knowledge of its origin.

MORTALITY.

Operation was performed in 37 of the 46 cases, with a mortality of 27 per cent. Of the 9 patients not operated on, 8 died and 1 recovered. Eight patients died on the table or within forty-eight hours of operation from hæmorrhage and shock; 1 died on the seventh day from peritonitis and 1 from bronchopneumonia on the third day. These figures compare well with those recorded for traumatic rupture of the spleen in general. Thus Bessel Hagen, in 1900, reported 37 cases with 20 recoveries and 17 deaths; Lotsch, in 1908, reported 138 cases with a mortality of 37·7 per cent; and Planson, in 1909, reported 140 cases with a mortality of 37·1 per cent. Buxton, in 1922, found the mortality for all cases up to that time to be 28·8 per cent. In 220 cases in which operation was not performed, Berger noted a mortality of 92·3 per cent. Watkins found the mortality to be 100 per cent. The chance of recovery without operation is clearly exceedingly slight.

Certain factors are involved in the comparatively low operative mortality of 27 per cent. First, few patients had associated injury such as rupture of the stomach or kidney, and, second, many of these patients were under good medical care when secondary hæmorrhage took place. The influence of associated injuries may be judged from the fact that of the 9 patients with fracture of the ribs or rupture of the kidney 7 died as a direct result of the secondary splenic hæmorrhage.

SUMMARY.

1. A case is described in which delayed hæmorrhage from the spleen occurred nine days after the original splenic injury.

2. Forty-five similar cases have been collected from the literature. Clinical study shows that the entire group presents a well-defined clinical syndrome, following the initial hæmorrhage and during the latent period, which should make the diagnosis possible before secondary hæmorrhage takes place.

3. Pathologically, the primary splenic injury consists of three varieties of lesion, the hæmorrhage from which is temporarily or permanently localized by various agencies.

4. The latent period is occupied by attempts at healing on the part of the spleen, and is usually marked by distinctive clinical symptoms.

5. Secondary hæmorrhage takes place in a high proportion of these minor splenic injuries, and is then associated with the same mortality as for splenic rupture in general.

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MALIGNANT GIANT-CELL TUMOUR OF BONE.

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TUMOURS of bone comprise a group of neoplasms whose many problems, pathological and surgical, are generally appreciated and require no emphasis. Standing out in sharp relief from them owing to even greater difficulties of interpretation, are the giant-cell tumours, particularly the ' benign giant-cell tumour '.

The difficulties surrounding this growth are exemplified by the numerous ideas expressed in the literature as to its origin as well as by the various changes in the general opinion as to its nature during ninety years. Various conceptions of the origin and nature of the benign giant-cell tumour have been expressed in a varied terminology—' myeloid sarcoma ',[28] ' giant-cell sarcoma ',[2] ' tumeur à myéloplaxes ', ' osteoclastoma ',[11, 13, 23] ' myeloma ',[29] ' myeloid tumour ',[7] and ' chronic (non-suppurative) hemorrhagic osteo-myelitis '[4] amongst others.

Originally they were regarded as malignant, probably owing to their confusion with the frank bone sarcomata. Their relatively innocent nature was then emphasized by several authorities and for several decades their relative innocence was accepted. Now, however, opinion is veering to an intermediate position, since during the last few years a considerable number of malignant examples have been described. It is the malignant growth which is to be considered here, with special reference to its differentiation from the innocent form.

HISTORICAL.

The giant-cell tumour was described first in 1845 by Lebert. It was further discussed in 1854 by Sir James Paget, who emphasized its benign nature, though stating, " cases may be found . . . in which a malignant course is run ". In 1860 Nélaton again expressed the belief that the growths were only locally malignant. This opinion became firmly entrenched in pathological and surgical writings. It may be exemplified by such papers as that of Kramer.[19]

During the last twenty years the controversy as to whether the tumours were really neoplasms or chronic inflammatory conditions has waged. The relationship of certain of these tumours to osteitis fibrosa cystica was also remarked. This dispute is still far from being settled, but a still more important problem has arisen.

In 1923 Stone and Ewing[30] and Turner and Waugh[21] reported examples of ' benign ' tumours which showed definitely malignant characters, though

the former considered that their case was an example of a malignant tumour of bone (osteogenic sarcoma) supervening on the innocent growth.

In 1924 Coley[9] reviewed a series of cases and demonstrated that not only are many of such cases malignant, but that the differentiation of the innocent from the malignant may be extremely difficult. He reported further cases in 1927.[10] In 1924 MacGuire and McWhorter[20] reported, in a series of twenty giant-cell growths, four malignant examples. Other cases were reported by Finch and Gleave[13] in 1926 and by Chatterton and Flagstad[8] in 1927. The case of Finch and Gleave developed pulmonary metastases.

In 1929 Sosman[27] pointed out a radiological peculiarity in malignant examples. In 1931 Dyke[11] and Orr[3] referred to malignancy in what were originally thought to be benign tumours. Last year also Simmons[26] reviewed 116 cases of 'benign giant-cell tumour' from the Registry of Bone Sarcoma of the American College of Surgeons.

The important feature of all these reports is the apparent impracticability of separating innocent from malignant growths except by means of their clinical course. Since the understanding of abstruse and border-line cases may best be achieved by the appreciation first of the characters of typical cases, this example is given.

CASE REPORT.

J. O., male, age 56. Sought treatment (in 1928) for a 'lump' in his right arm of four years' duration. Except that he had generalized psoriasis, he had had no disabilities. He had had a fall on to his left arm five years before coming for advice, and some time after this he noticed a swelling on the outer side of the lower part of the forearm. This remained stationary in size until two months before he sought attention, when it began to be painful and to enlarge.

ON EXAMINATION.—There was a swelling about 2 in. in diameter occupying the region of the lower end of the radius. It was hard, not tender, and there was no egg-shell crackling.

X-ray Examination.—The appearance was typical of 'benign giant-cell tumour' of bone. There was concentric enlargement of the bone, thinning of the cortex, though without loss of continuity, and the characteristic trabeculation of the substance of the tumour. The patient was treated by deep X-ray therapy. The increase in size continued and the pain was not relieved.

Five months after first coming for treatment he was admitted to hospital for operation. At this time X rays showed that the condition was similar to that seen previously, but there was loss of continuity of the cortex in several places and there was obvious invasion of the soft tissues by the tumour (Fig. 106). The significance of this finding, at that time, was not appreciated.

OPERATION.—This consisted of removal of the lower quarter of the radius with the introduction of a bone-graft.

PATHOLOGICAL EXAMINATION.—Microscopically the tissue consisted of a large number of foreign-body giant-cells of the typical 'myeloid' type (Fig. 107), a cellular stroma containing a large proportion of spindle cells, and considerable hæmorrhage. A diagnosis of benign giant-cell tumour was made.

Later, when subsequent events were an incentive to a review of the case, the examination of the remainder of the specimen revealed a very different picture. Many areas were extremely cellular, consisting largely of active spindle cells (Figs. 108, 109). Mitotic figures were present in numbers. In some parts a large number of giant cells of both tumour and foreign-body type were present. In some cases the tumour giant cells were in excess (Fig. 110). The tissue was very vascular. Destruction of bone was occurring at the growing edge.

FIG. 106.—Tumour of the left radius. The arrows indicate the site of breaking through the capsule and the extent of the tumour invading the soft tissues. The thickening of the bone and trabeculæ is probably the result of the radiation therapy. Previous films did not show these features, but were typically those of benign giant-cell tumour.
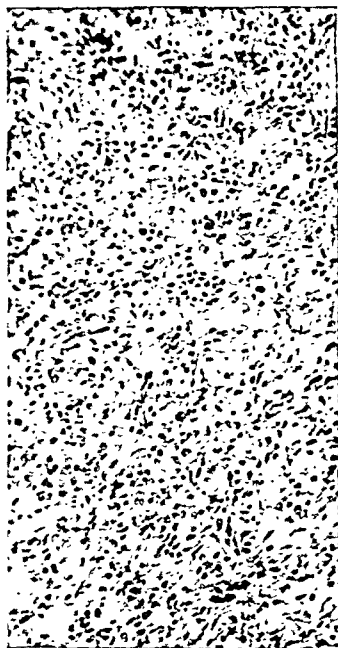
FIG. 107.—Section of the growth which was first examined and on which the diagnosis of benign giant-cell tumour was made. (× 80.)
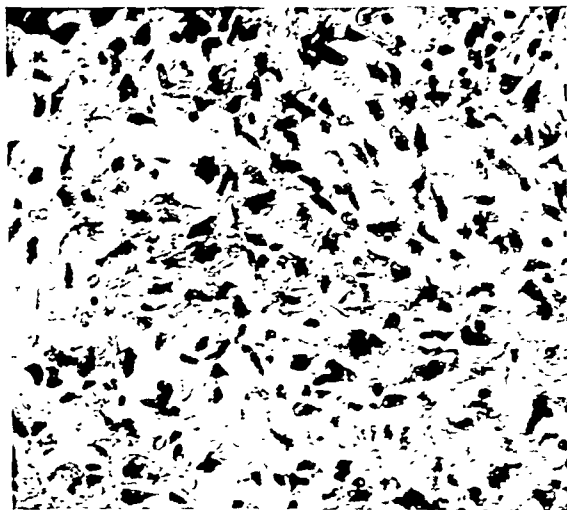
FIG. 108.—Portion of the section showing spindle cells and giant cells. (× 400.)

FURTHER OPERATIONS.—The patient progressed satisfactorily for a time. Six months later fracture occurred through the graft, and since the growth had recurred, amputation through the forearm was performed. Twelve months after amputation a nodule was found to have developed in front of the elbow (? enlarged epitrochlear gland), and this swelling rapidly increased until a large mass was present (*Fig.* 111).
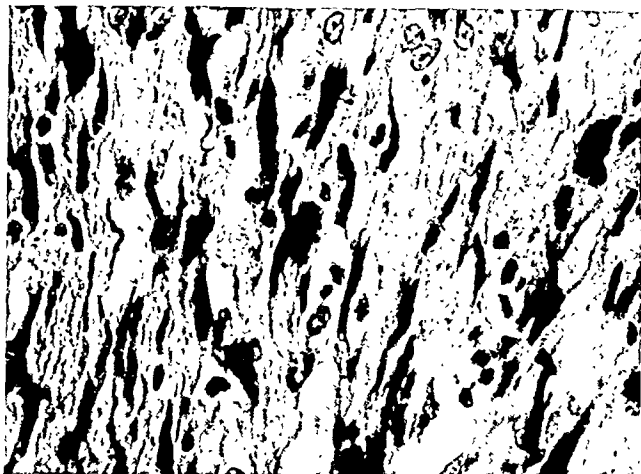
FIG. 109.—Another portion of the tissue showing active spindle cells, some of which are giant cells. (× 400.)

Amputation was performed through the neck of the humerus. No immediate complications resulted.

PATHOLOGICAL EXAMINATION.—The mass caused a fusiform swelling of the arm just above and involving the elbow (*Fig.* 112). It measured 5 in. in diameter and about 8 in. long. The tissue was soft and hæmorrhagic. At its edge it was invading the surrounding tissue.
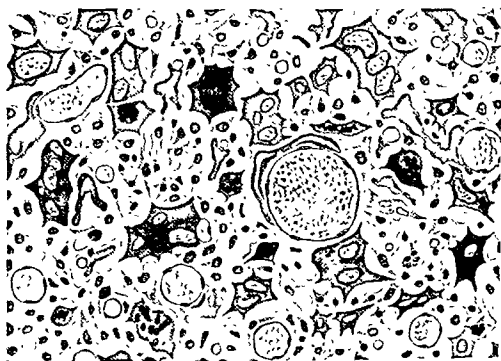
FIG. 110.—Section of another portion of the tumour. There are numerous giant cells, mitotic figures, and a few foreign-body giant cells (hæmatoxylin and Van Gieson).

Microscopically the tissue was very cellular, consisting of spindle, spheroidal, and many tumour giant cells (*Figs.* 113, 114). The tissue was extremely vascular. In some areas many cells were of the bone-marrow type.

Shortly after the second amputation the patient complained of anorexia, cough, disinclination for exertion, and loss of weight.

X-ray examination of the chest showed an appearance consistent with numerous secondary growths in both lungs (*Fig.* 115). There was also an area in the 1st lumbar vertebra suggesting a metastasis. Two months later the patient died suddenly. Unfortunately a post-mortem examination was not obtained.

This case is, in review, a typical example of a malignant giant-cell tumour* of bone and one which is readily distinguishable from the innocent form. It differs from the benign giant-cell tumour : (1) In presenting a peculiar radiological appearance ; (2) In its microscopical characters—tumour giant cells, mitotic figures, and a predominance of spindle cells ; and (3) In its clinical

FIG. 111.—The elbow region after the development of the secondary tumour in this region. The secondary growth which was thought on clinical grounds to be in the epitrochlear gland is here seen to be extra-osseous.

FIG. 112.—Photograph of the tumour in the region of the elbow after amputation through the shoulder. H, Humerus : E, Elbow-joint. The section does not pass through the humerus all the way.

course with metastasis formation. Two at least of these features however, (1) and (3), are late developments, and diagnosis before these occur, especially (3), is desirable. Simmons says, " In analysing these cases it is seen that there is no instance in which a proved giant-cell tumour may change its character and become osteogenic." Even if this is true—and accumulated evidence in the literature does not support the statement—it does not help

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* The term 'giant-cell tumour of bone' is used in this paper to refer only to the malignant form of the benign giant-cell tumour, and does not refer to obvious osteogenic sarcomata which contain giant cells.
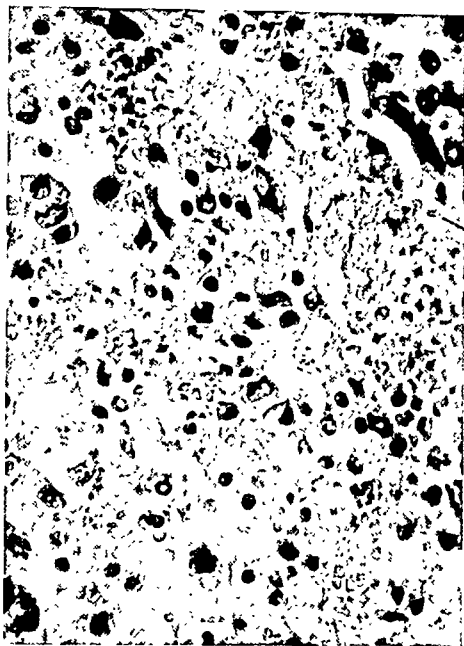
FIG. 113.—Section of the secondary growth in the arm showing the anaplastic and variable nature of the cells. The tissue is extremely vascular. (× 350).
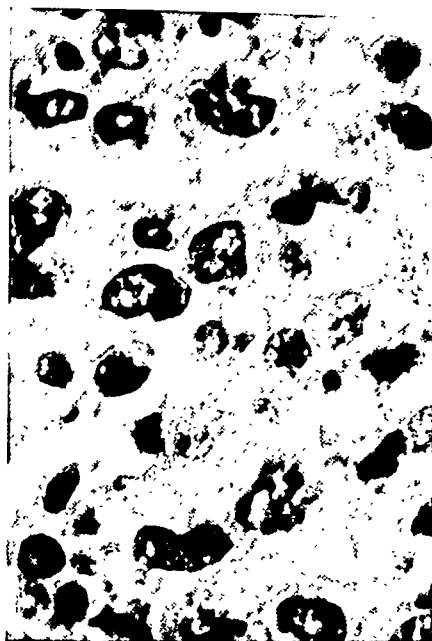
FIG. 114.—High-power photomicrograph of tissue similar to that shown in *Fig.* 113. The cells, which vary in size, contain large nuclei with dense chromatin. They form some tumour giant cells. (× 900.)
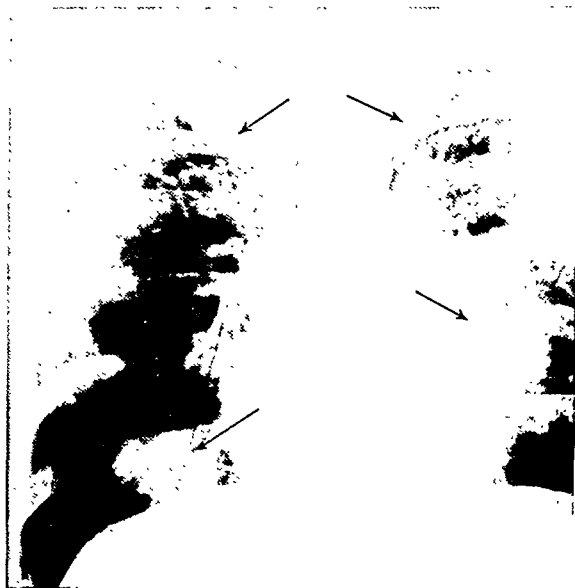
FIG. 115.—Radiograph of the chest obtained shortly after the amputation through the shoulder. The shadows thrown by the metastatic tumour masses are shown by arrows.

when, in the early stages of development of a growth, one is confronted with an X-ray picture typical of benign giant-cell tumour and a typical or almost typical microscopic picture.

A diagnosis of benign giant-cell tumour was made in the early stages in the case here described, but this was due to remediable errors—only one part of the growth was examined microscopically and even some of the features of this portion were overlooked, and the radiographs were incompletely interpreted. When the case was reviewed it was noticed that the tumour had invaded the surrounding tissues.

In 1919 Bloodgood[6] submitted evidence from which he considered it could be concluded that destruction of the bony capsule of the growth was no evidence of malignancy. His cases, however, do not allow of this conclusion, since in several amputation or radical removal of the growth was performed. That cure results from this treatment only means that these growths are not of the same order of malignancy as the osteogenic sarcomata. Though this invasion of the soft parts by the tumour described in this paper was an original observation at the time at which it was made, search of the literature subsequently showed that Sosman had also referred to it. It is well shown also in the illustrations of many of the cases described in the literature (e.g., see Bloodgood's[6] and Coley's[9] papers).

Most of the statements made concerning these tumours conclude with the opinion that differentiation between the innocent and malignant varieties is not possible. Simmons[26] concluded this from the study of the 116 cases in the Registry of Bone Sarcoma. Coley[9] obtained opinions from a number of well-known pathologists and received diametrically opposed opinions from some of them on the same or similar material. The inference that differentiation is therefore impossible is, however, quite unreasonable. All that we can deduce is that, in the present state of our knowledge, diagnosis is extremely difficult. In the final analysis, malignancy or innocence may be determined only by the life-history of the tumour, and the microscopic appearance must be correlated with this. Prognosis can only be argued (with any degree of certainty) from the microscopic appearances after certain microscopic findings had been observed to be associated with a definite clinical course over a large series of cases. This somewhat obvious even if not axiomatic statement is, I think, not sufficiently appreciated. Our present knowledge of bone sarcomata is not complete enough to allow of the accuracy of diagnosis and prognosis which is possible with—for example—some of the carcinomata and other tumours.

Are there any criteria at all that will aid in the diagnosis of these tumours ? There are three possible sources of information, the clinical observations, pathological investigations, and radiological examination.

1. There do not appear to be any criteria of differentiation of malignant from innocent giant-cell growths by clinical investigation in the early stages. The development of metastases as a mode of differentiation is unsatisfactory, to say the least, from the patient's point of view.

2. So many errors have been made during the last few years in the microscopic diagnosis of the malignant forms of the giant-cell growths that at first sight the opinion expressed by several writers seems almost justified.

However, a few features are worth consideration. In the typical example there is a predominance of spindle-cell tissue, the presence of mitotic figures, tumour giant cells. The typical picture of the benign growth, on the other hand, is a polymorphic tissue containing round and spindle cells, foreign-body giant cells, and hæmorrhagic and degenerative foci.

These two forms may be intermingled, and such cases are those which present the difficulties. It has been pointed out by several writers, and particularly by Ewing, that injury, hæmorrhage or infection, or bone destruction in a tumour of bone may result in the formation of giant cells surrounded by a cellular stroma of pleomorphic cells. The significance of this observation is that the foreign-body giant cells must be ignored in making a diagnosis and an attempt made to disentangle the true stroma of the tumour from the various inflammatory cells which may be intermingled with them.

That irritation (mechanical, chemical, or bacterial) may give rise to a change in type of a benign giant-cell tumour is stated by several authorities. The evidence for this opinion seems to be good, though by no means complete. Malignancy undoubtedly develops in a number of cases which are submitted to trauma. These are the very cases in which the microscopic picture is most confused and most closely approximates to that of the benign giant-cell growth. In some areas it may be identical.

This may be exemplified by one of several examples : I was asked to examine a piece of tissue which had been removed from a tumour in the upper end of the tibia. It was thought, clinically, to be a benign giant-cell tumour. Microscopic examination showed a tissue consisting of spindle cells mainly, with one mitotic figure in about two lower-power fields. There were numerous foreign-body giant cells and no tumour giant cells. The report of a malignant tumour was made. Other opinions of an opposite nature were obtained by the surgeon in charge of the case, and he was satisfied with curettage. Three months later the tumour recurred and fungated through the wound. A fresh piece of tissue on microscopic examination presented an even greater resemblance to the benign giant-cell tumour, the foreign-body giant cells being even more abundant and the stroma even more pleomorphic. This was then used as evidence for the conclusion that it was not possible, microscopically, to differentiate malignant from innocent growths, although areas in which spindle-cell tissue predominated and where mitotic figures were present were found. Such a conclusion, however, ignores the relationship of the excess foreign-body giant cells and inflammatory cells in the stroma with hæmorrhage or infection of the growth. The patient died later from pulmonary metastases.

I would state, therefore, that with careful examination of the tissue and the correct stress on the various parts of the tissue it should be possible in many cases at least to determine that the growth is malignant. I have found the most valuable criterion to be the presence of an occasional mitotic figure. The search may resemble that for tubercle bacilli in a section stained specially for them. When one is found, however, others are usually also quickly discovered. Later others or more satisfactory criteria may also be found, and a search for them is necessary before drawing the conclusion that they are completely absent.

That different parts of a tumour may show very different morphological appearances requires special attention. If hæmorrhage or infection involves one portion of a growth more than another, then in all probability—as suggested above—such areas will more closely resemble the benign form of growth. This should be remembered also when pieces of tissue are obtained for microscopic examination.

3. Radiological investigation in the early stages may throw no light on the prognosis of the growth. Before long, however, some variations in appearance may be found, particularly loss of continuity of the covering bone and invasion by tumour mass of the surrounding soft tissues (*see Fig.* 106). The trabeculation may become less regular than is usual. It is necessary therefore to repeat the radiological examination of the growth at intervals and watch particularly for such changes.

During the last few years I have encountered six cases in which this question of type and malignancy of the growth has arisen and which proved to be malignant. An erroneous conclusion was drawn in two of these, but in retrospect this was due to non-observance of the conclusions drawn here— for example, in one case only one small piece of tissue led to a diagnosis which, even now, on that particular slide seems justified. This was taken from a fungating piece of growth which was infected. In another case the (then) doubtful microscopic picture was considered to be subservient to the clinical history and the radiological appearances.

In view of the observation of Simmons that 7·5 per cent of the ' benign giant tumours ' treated conservatively give rise to pulmonary metastases, it is necessary to examine the tumours as carefully and fully as possible, make repeated radiological examinations, and accept any one criterion of malignancy even if not supported by other findings and even if this is found only in one part of the growth.

On the first evidence of malignancy the growth should be removed. It has been shown that complete removal of these tumours in an early stage of development or, according to some authorities, before the growth has changed to an osteogenic sarcoma, will prevent the formation of metastases.

SUMMARY.

An example of a malignant form of giant-cell tumour of bone is described. The means by which the malignant growth may be distinguished from the innocent form are discussed.

Microscopically the stroma is cellular, composed mainly of spindle cells with active nuclei and an occasional mitotic figure. Tumour giant cells, when present, are very characteristic. Foreign-body giant cells and pleomorphic stroma, so characteristic of the innocent ' growth ', must be disregarded when determining prognosis, since they may predominate if degeneration, infection, or bone destruction is present in the area under examination. In order to minimize this difficulty specimens should be taken where possible from areas which do not show these complications.

In the later stages of development of the growth there is a characteristic radiological appearance. Thus it is necessary in a doubtful case to make

radiographic examinations at intervals in order to observe the earliest sign of invasion of the bone cortex or the surrounding tissues. Several portions of the growth should if possible (e.g., when curettage is performed) be examined. Positive evidence of any kind, even if found in only one part, should be accepted to the exclusion of negative evidence.

I would thank Mr. A. F. Maclure, F.R.C.S., for the notes of the case, and Dr. L. A. Love for assistance in the elucidation of the radiographs.

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THE RELATIONSHIP BETWEEN PEPTIC ULCERATION AND GASTRIC CARCINOMA.*

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HISTORICAL INTRODUCTION.

THE history of this problem commenced with Cruveilhier, who in 1839, in his *Atlas of Pathological Anatomy*, was the first to distinguish clearly between chronic ulcer and carcinoma both clinically and pathologically. He also discussed the association of the two conditions and finally says : " La question de savoir si un ulcère chronique simple peut devenir cancéreux ne me parait devoir être résolue d'une manière affirmative que pour les individus affectés de la diathèse cancéreuse."

A few years later (1842) Rokitansky stated that cancer sometimes occurred with simple ulcer and that it might arise from the ulcer. Dittrich, in 1848, collected 160 cases of cancer of the stomach and found 6 developing near ulcers ; in 2 of these the carcinoma was limited to the edge of the ulcer. He appears to have regarded this as a coincidence rather than a causal factor. Brinton, in 1858, published a full description of " a case of cancerous infiltration of a chronic gastric ulcer." It was in the middle third of the lesser curvature and eroded the liver. Its floor and edges were cancerous. He had little doubt that it was a gastric ulcer which had undergone a cancerous infiltration or deposit because of : (1) Long duration ; (2) Absence of cachexia as well as secondary deposit ; (3) Healthiness of the gastric tissues around ; (4) Exact coincidence of deposit and ulcer. Later (1864) he discusses the question more fully and seems to think the combination of ulcer and cancer not a mere coincidence. He also mentions the question of peptic ulceration of growths and considers the complete destruction of a tumour by peptic digestion to be possible in rare instances.

In 1868 two theses on this subject were presented to Berlin University by Steiner and by Wollmann. Both estimated the proportion of cases of carcinoma arising in ulcer at 4 per cent, but neither produced any morbid anatomical proof of their statements. Mayer (1874) published a case of carcinoma of the stomach which macroscopically resembled a callous ulcer, but on histological examination showed islets of carcinoma in it. This appears to be the first recorded histological examination of an ulcer-cancer. These three papers were well received in Germany and were the means of popularizing the idea that gastric ulcer was at any rate sometimes followed by carcinoma.

* A thesis for which the Raymond Horton Smith Prize of the University of Cambridge was awarded in 1931.

They seemed to have caused Lebert to alter his views, for in 1861 he said that cancerous degeneration in simple ulcers is possible but has not yet been demonstrated, while in 1878 he estimates that 9 per cent of gastric cancers are produced in this way.

Four years later Hauser (1882) described a case of ulcer-cancer which had been found accidentally at autopsy on a man dying of pneumonia with no history of previous indigestion. He also emphasized its importance in the etiology of gastric cancer in general. Discussing this paper, Zenker expressed the opinion that most cases of carcinoma of the stomach arise in pre-existing peptic ulcer.* Hauser published several papers, and in 1890 a book on carcinoma of the stomach and intestine in which four cases of ulcer-cancer were described and one was illustrated. This illustration has been copied many times—notably in Ewing's text-book—and remains the classical example of ulcer-cancer.

Duplant, in 1898, attacked these views in his thesis at Lyons. After outlining the previous work on the subject, he describes fully eight cases of supposed ulcer-cancer and concludes that they were " en réalité de néoplasmes ulcérés." He suggested that one type of carcinoma is similar to the rodent ulcer of the face, and when ulcerated closely resembles simple peptic ulcer. He quotes Tripier and Bard as having seen cases where the primary carcinoma has ulcerated away and the essential malignant nature of the ulcer could only be diagnosed by the presence of metastases. Finally he says that his observations make him believe that the grafting of carcinoma on ulcer is impossible.

Similar views that ulceration of primary carcinoma is more common than cancerization of an ulcer, have been put forward by Stromeyer (1912) and others from Aschoff's school, Moskowicz (1924), and many others.

The chief exponents of the opposite view, that most cases of carcinoma arise in pre-existing peptic ulcer, have been the surgeons and pathologists of the Mayo Clinic. Starting with W. J. Mayo in 1907, a long series of papers has been published by McCarty, Broders, Wilson, and others, all expressing the view that 50 to 70 per cent of gastric cancers arise in ulcers.

Very many papers have been written on this subject, and the way in which opinions have varied is shown in *Figs.* 116, 117. *Fig.* 116, copied from Cabot and Adie (1925), shows the way the pendulum of opinion has swung

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* This opinion of Zenker's seems to have been exaggerated by many later writers. For instance, Duplant (1898) says : " Zenker, dans un enthousiasme qui n'a rien de germanique, n'hésite pas à déclarer que tous les néoplasmes de l'estomac ont l'ulcus comme point de départ ", and gives the reference (*Berl. klin. Woch.*, 1882). Audistère (1903) also quotes the same reference and says that for Zenker " tous les cancers de l'estomac se développent sur les ulcères simples." Cabot and Adie (1925) make the statement that Zenker " proposed the idea that all gastric carcinoma originated in ulcer ", and Stewart (1929) remarks, " The swing of the pendulum reached its height with Zenker (1882), who expressed the opinion that most if not all cancers have their origin in simple ulcer." Several other writers make similar statements, and the only reference given to support them is that from *Berl. klin. Woch.*, 1882, xix, 657. This turns out to be the report of a meeting of a congress at Eisenach, where Zenker congratulated his assistant Hauser on his paper " und spricht die Meinung aus, dass auch aus dem klinischen Verlaufe vieler, ja der meisten Fälle von Magencarcinom auf ein vorhergegangenes Magengeschwür zu schliessen sei." Unfortunately it has not been possible to obtain a verbatim report of Zenker's remarks or to find any other reference. It is difficult to understand how ' meisten Fälle' has been translated as ' all' or ' 100 per cent.'

between 1845 and 1925. *Fig.* 117 is a graph in which the number of reports expressing similar percentage figures of the frequency of ulcer-cancer is plotted against the percentage figure given. A similar graph is given by Cabot and Adie, but as it is not drawn to scale and no references as to the source of the figures are given. it has been neglected. The source of the figures in *Fig.* 117 is given on p. 305. The total number of reports collected is 102, and it will be noticed that 51, or one-half, estimate that not more than 10 per cent of gastric carcinoma arise in ulcers ; 74 believe it is less than 20, and only 15 give an estimate over 50 per cent.

FIG. 116.—Variation of opinion between 1845 and 1925 of number of gastric cancers arising in ulcers. (*From Cabot and Adie.*)
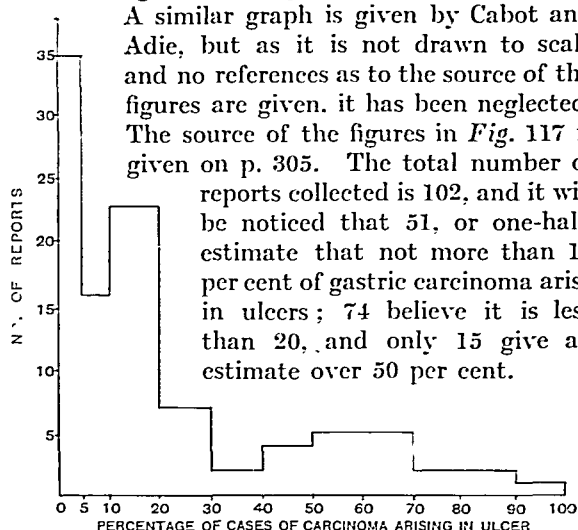
FIG. 117.—Graph showing number of reports expressing similar percentage figures of the frequency of ulcer-cancer in gastric carcinoma.

MATERIAL.

The material studied for the purpose of this paper consists of all the portions of stomach from surgical operations received in the Department of Pathology, St. Mary's Hospital, Paddington, from January, 1920, to April, 1931, inclusive. Most of them have come from cases in hospital, but a few have been sent from outside. In all, 307 stomachs were received, and their various lesions are shown in *Table I.*

Table I.—VARIOUS LESIONS IN 307 STOMACHS.

Simple chronic gastric ulcer	154
Gastric carcinoma	46
Duodenal ulcer	75
Jejunal ulcer	7
Subacute gastric ulcer	4
Other cases omitted	22
Total	**307***

* These figures add up to 308 as in one case (576125) both ulcer and cancer were present although separated (*see* p. 302)

The cases omitted were :—

Too little received for diagnosis	10
Gastrojejunostomy junctions	3
Gastritis	2
Hypertrophied pylorus	2
Myoma	2
Sarcoma	1
Linitis plastica (inflammatory)	1
Carcinoma of œsophagus	1

To form a control group 112 consecutive operation cases of carcinoma of intestine were studied also. Post-mortem material has not been used for statistical purposes, although it has been used in certain cases to amplify observations made with the surgical material. In order to have clear ideas on the relationship between peptic ulcers and gastric carcinoma it will be necessary to consider briefly the morbid anatomy and histology of these conditions.

MORBID ANATOMY AND HISTOLOGY OF PEPTIC ULCERS.

Peptic ulcers, as their name implies, are the result of digestion of the stomach or intestinal wall by gastric juice. The factor or factors necessary to allow this digestion to take place, be they inflammatory, nervous, ischæmic, or otherwise, do not concern us now. We are only interested in their morbid anatomy and histology. They are usually divided into two groups—acute and chronic. It is only the latter which need be considered here ; but it is now generally recognized, following the work of Bolton and others, that the so-called chronic ulcer arises from an acute ulcer in which healing is delayed—in fact, peptic ulcers are not chronic in the sense that tuberculous and syphilitic lesions are chronic, but rather represent the balance at any one time between successive attacks of acute digestion alternating with periods of healing. The digestive action, whenever it takes place, is an acute process ; between the active attacks repair is taking place. The appearance of an ulcer, therefore, will vary greatly according to the phase of the attack in which it is removed and also the number of attacks which have occurred.
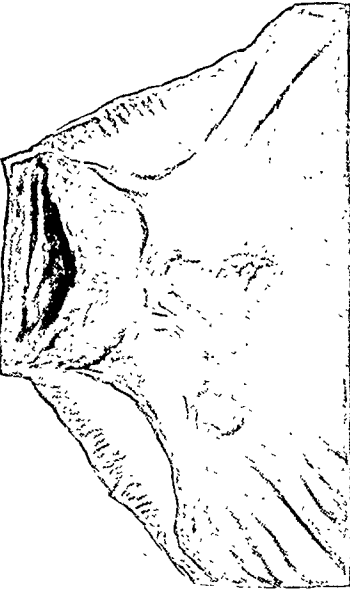
FIG. 118.—Multiple peptic ulcers in stomach. Pyloric end of stomach and beginning of duodenum showing two chronic gastric ulcers in pyloric canal. The larger is on the lesser curvature and the smaller on the posterior wall. (× ⅔.)

Number. — Acute ulcers are frequently multiple, chronic are usually single. *Table II* shows the multiple ulcers in this series. (*Fig.* 118.)

Table II.—MULTIPLE PEPTIC ULCERS.

	Cases
Two chronic gastric ulcers	7
Two chronic gastric ulcers and chronic duodenal ..	1
Chronic gastric and chronic duodenal	3
Chronic gastric and acute duodenal	I
Total	12 in 154 (7·8 per cent)

Of the duodenal ulcers, 18 out of 75 (24 per cent) were multiple.

Position.— The position of chronic gastric ulcers is to a large extent restricted to the neighbourhood of the lesser curvature (*see Fig.* 121) and the

pyloric canal, the so-called 'Magenstrasse' or gastric pathway. The position of the cases in this series is shown in *Table III*, Professor Stewart's figures being added for comparison.

Table III.—SHOWING POSITION OF ULCERS.

POSITION		NUMBER	PERCENTAGE	PROF. STEWART'S PERCENTAGE	
				Surgical	P.M.
Lesser curvature	..	135	83·8	96·5	82
Pyloric canal ..	..	11	6·8 }	3·5	12
Pylorus	..	14	8·6 }		
Cardia	..	—	—	—	1
Anterior wall ..	..	—	—	—	2
Posterior wall ..	..	1	0·6	—	3

' Lesser curvature ' includes all ulcers touching or within 2 cm. of the lesser curvature. Under ' pylorus ' are included those actually involving the pyloric sphincter. Except in the case of one ulcer which had duodenal mucosa on one side and gastric on the other, all these ulcers were definitely on the gastric side of the pylorus, having gastric mucosa on both sides. These observations are in agreement with Moynihan (1923) that an ulcer is either duodenal or gastric, but do not agree with his further statement that a gastric ulcer is rarely within an inch and a half of the pylorus.

Shape.—The shape of the ulcers varies greatly. The smaller ones are generally more or less circular, the larger oval with the long axis in the long

FIG. 119.—Active chronic gastric ulcer. This is in an active phase and shows the 'layers' in base. The shape with overhanging cardiac edge and shelving pyloric edge is characteristic. P and C point to the pyloric and cardiac end respectively. (× 3·6.)

axis of the stomach or else saddle-shaped riding over the lesser curvature. They are irregular, usually more or less terraced or shelving in their edges, and a very frequent appearance is for the edge on the pyloric side to slope gently downwards while that on the cardiac side is steep or overhanging (*Figs.* 119, 120).

Size.—The ulcers vary greatly in size. The largest in this series was 3 cm. diameter, the majority being about 0·5 cm. It has been stated that ulcers above a certain size, generally given as 1 in. in diameter, are malignant.
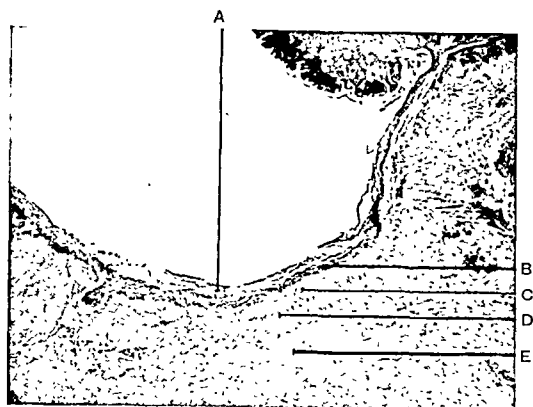
FIG. 120.—Cardiac side of the ulcer in *Fig.* 119 under higher magnification to show 'layers'. A, Mucus with a few leucocytes ; B, Fibrin and leuco- cytes ; C. Necrotic layer ; D, Granulation tissue ; E, Fibrous zone. (× 10.)

FIG. 121.—A large peptic ulcer with an eroded vessel in its base. It is on the lesser curvature, P and C pointing to pylorus and cardiac end respectively. (× ⅜.)

This is, of course, quite untrue. Size has nothing to do with malignancy. A large ulcer removed post mortem is shown in *Fig.* 121. The largest I have seen in another post-mortem specimen was 9·5 cm. × 5·5 cm., but many larger have been recorded.

Histology. — The histology of ulcers naturally varies according to the phase in which they are seen. Since Askanazy (1920) published his classical account of chronic peptic ulcer, it has been customary to describe the floor of the ulcer as having several zones or layers in the active phase of ulceration (*Fig.* 120).

1. The surface is often covered by mucus in which a few leucocytes are entangled.

2. A fibrinous layer containing leucocytes. This layer is most abundant in very active ulcers, when it usually contains many neutrophil polymorpho- nuclear leucocytes. In very early ulcers, or those with a recent exacerbation, the leucocytes may be scanty.

3. A necrotic layer permeated with fibrin. It is usually nearly structure- less, but may contain the 'ghosts' of arteries or nerves.

4. A zone of granulation tissue generally sharply separated from the necrotic layer above but gradually blending into the fibrous zone beneath. It is made up of fibroblasts which are forming collagen fibre and capillary loops which are not often very numerous. This zone is always infiltrated by leucocytes : eosinophils, plasma cells, and lymphocytes being the most numerous. Neutrophil polymorphs are usually absent.

5. A dense fibrous zone. This is not sharply demarcated from the zone above it and spreads beyond the margins of the ulcer at its edges. In some cases it is remarkably vascular and in others vessels may be scarce. It sends

processes into and often contains nodules of the fatty tissue of the lesser omentum.

Margins.—The margins of the active chronic ulcer vary considerably. Where active digestion is taking place they are usually sharply defined, but may overhang, especially on the cardiac side of the ulcer. In this way the mucosa and muscularis mucosæ may be bent down towards the base of the ulcer. When healing is taking place the edges become more shelving ; this is usually seen especially on the pyloric side.

Muscular Coat.—This is usually destroyed in chronic ulcers, and was in all but five of those in this series. In the active phase the muscular fibres may stop abruptly at the edges of the ulcer, but more usually they are separated by œdema, granulation tissue or fibrosis spreading between the fibres. In this way the muscular tissue is generally spread out fanwise at the edge of the ulcer. As the process of healing progresses the overhanging muscularis mucosæ and the spread-out fibres of the muscularis become approximated and eventually fuse. This close approximation or fusion of the muscularis and muscularis mucosæ was present in some part of all but two ulcers in this series. (*Figs.* 122, 123.)
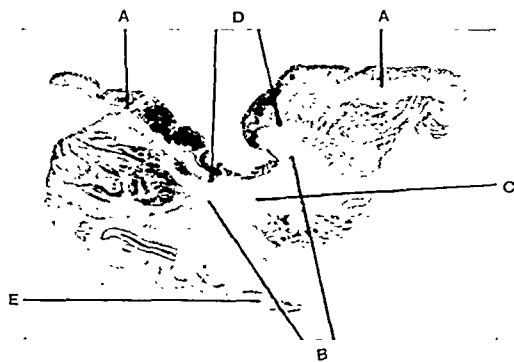
FIG. 122.—Healing peptic ulcer of stomach.
A, Muscularis mucosæ ; B, Area in which muscularis is destroyed ; C, Area of dense fibrosis ; D, Points of fusion between muscularis mucosæ and muscularis · E, Vessel with endarteritis. (× 3.)

FIG. 123.—The cardiac side of the ulcer in *Fig.* 122 with higher magnification to show the muscularis mucosæ curling round and fusing with muscularis. A, Muscularis mucosæ ; D, Point of fusion between muscularis mucosæ and muscularis. (× 10.)

Hauser (1895) called attention to the way in which the muscular coat is frequently turned up into the floor of the ulcer owing to the contraction of the cicatricial tissue formed in the subserous region and in the omentum. This is frequently present, but by no means peculiar to peptic ulcers ; in fact, the contraction of similar subserous fibrous tissue in carcinomata of the large intestine is often the cause of the tight annular constriction seen in these cases.

Blood-vessels.—The blood-vessels in and near an ulcer are frequently the site of pathological changes. Obliterative endarteritis, phlebitis and thrombosis, sometimes with recanalization, are the changes most frequently seen.

Healing (*Figs.* 124–126).—This commences with the separation of the slough formed of the necrotic tissue and fibrin. The granulation tissue layer becomes organized and the newly formed fibrous tissue contracts. At the same time the epithelium at the edges grows in over the surface of the granulation tissue as a single layer of cubical or flattened cells. These go on proliferating and send small downgrowths into the granulation tissue, which at the same time pushes up little papillary processes. These processes become vascularized and the cells covering them often become tall and columnar. In this way a new mucosa is formed. It never returns to the normal, but is always thinner. The glands are fewer and are lined by a lower type of epithelium.

FIG. 124.—Chronic gastric ulcer healing in some parts and extending in others. (× 3.)

This regeneration of mucosa implies active growth of epithelial cells. If at the same time the newly formed fibrous tissue on which the epithelium is
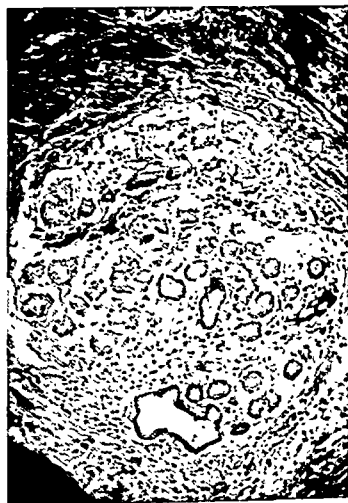
FIG. 125.—The left side of the ulcer in *Fig.* 124 with higher magnification. At the edge of the main ulcer the epithelium is growing in and forming large papillæ. Quite near to the healing area is a small secondary crater where digestion has recently been active. Further away from the ulcer in the fibrous zone there is an area of deep epithelial heterotopia (A). (× 12.)

FIG. 126.—The area of heterotopic epithelium seen in *Fig.* 125 under higher magnification to show the regular cells of low cubical shape. This is not carcinoma. (× 120.)

resting contracts, it will draw down actively growing epithelium into the deeper tissues. In this way regenerating epithelium at the edge of ulcers is frequently misplaced and may be seen situated deeply in the submucosa or even in the muscularis. This is a frequent occurrence in healing ulcers and was present in 44 of 161 ulcers (29 per cent) in this series.

MORBID ANATOMY AND HISTOLOGY OF GASTRIC CARCINOMA.

Number.—Carcinomatous lesions are nearly always solitary, and in this series there is no example of multiple carcinomata.

Position.—The position of the lesions is shown in *Table IV*, to which again Professor Stewart's figures have been added for comparison.

Table IV.—POSITION OF LESION IN GASTRIC CARCINOMA.

POSITION		NUMBER	PERCENTAGE	PROF. STEWART'S PERCENTAGE	
				Surgical	P.M.
Pyloric canal ..	..	23	57·5	88·7	66·5
Cardiac end ..	..	—	—	—	15·2
Lesser curvature	..	8	20	7·5	12·8
Rest of stomach	..	9	22·5	3·8	5·5

Size and Shape.—Gastric carcinoma may roughly be divided into three groups :—

1. Vegetating or fungating cases (*Fig.* 127) which grow mainly into the lumen of the stomach and form large cauliflower-like masses. Eight examples of this type occurred in this series. The smallest was 3·5 cm. in. diameter,

FIG. 127.—Fungating carcinoma of pyloric end of stomach. (× ⅔.)

and the largest 9·5 cm. Post-mortem specimens up to 30 cm. in diameter at one time were not uncommon.

2. Leather-bottle stomach or linitis plastica (*Fig.* 128), a condition in which the growth rapidly infiltrates a large area of stomach wall. The site of the starting-point of the growth can usually be seen as a small ulcerated area,
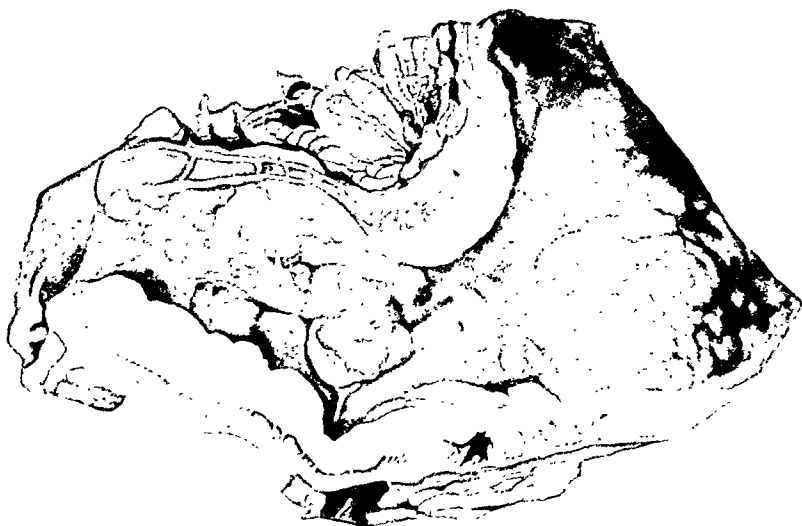
FIG. 128.—Half a leather-bottle stomach removed at operation. (× ⅔.)
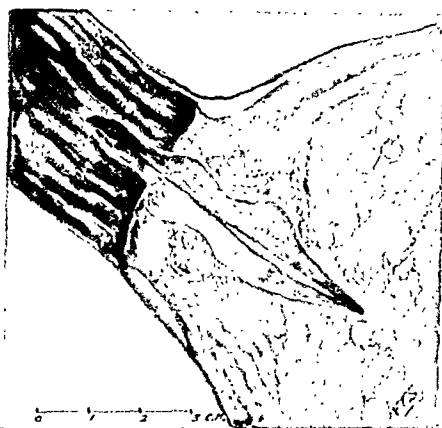
FIG. 129.—Ulcerated carcinoma of stomach. A portion of stomach and duodenum opened along the lesser curvature to show an ulcerated carcinoma on the greater curvature. (× ⅔.)

generally at the pyloric end. The growth usually spreads widely in the submucous and muscular coats. Three examples occur in this series, and as in each case the growth extended up to the edge of the portion removed, it is impossible to estimate their sizes.

3. *Ulcerated infiltrating cancer (Figs.* 129, 130). This group is the most important for the purposes of this paper, as these are the cases often thought to arise in peptic ulcers. Thirty-three were present in this series. The ulcerated carcinoma has a crater which may penetrate to any depth or even perforate the stomach wall. Its edges are usually raised and irregular, and the base is hard. In size they vary considerably. The smallest in my series was 1·5 cm. in diameter across the ulcer, while the largest was 4·2 cm.

Examination of a cut surface or section with a hand lens will usually make clear several features. The carcinoma at the base of the ulcer is generally spreading centrifugally in all directions;

FIG. 130.—An ulcerated carcinoma of stomach. A, Muscularis mucosæ thrown up by carcinoma in submucosa; B, Carcinoma in muscularis between but not destroying the muscle fibres. (×2.)

especially it tends to grow into the loose connective tissue of the submucous coat, and so separate the muscularis mucosæ from the muscularis. Typically the growth can be seen invading the muscularis running between the muscle fibres but not destroying them completely. If the growth has been present for some time, the muscle may be completely destroyed in the centre, but usually a few muscle fibres can be made out. The amount of fibrous tissue formed will vary according to the type of growth—scirrhous or medullary. As a general rule, large areas of fibrous tissue free from growth are not produced.

Histology. — The finer histology of carcinoma does not affect the argument of this paper, and may be briefly dismissed. Most commonly the growth is arranged in alveoli, tubules, or papillæ lined or covered by columnar cells. Sometimes the growth is almost entirely undifferentiated, consisting of solid masses and trabeculæ of polygonal cells. Occasionally, as in the leather-bottle type, the cells are so few and arranged in such small groups that their epithelial nature may be difficult to determine.

HISTOLOGICAL CRITERIA FOR THE DIAGNOSIS OF ULCER-CANCER.

The problem is twofold :—

1. **The Diagnosis of the Pre-existence of Simple Ulcer.** — There are four points in which a simple ulcer usually differs from carcinoma in the stomach : (*a*) Complete destruction of an area of muscle corresponding in size roughly to the floor of the ulcer ; (*b*) The presence of a large area of dense

fibrous and granulation tissue in the floor of the lesion; (c) The presence of endarteritis obliterans or thrombophlebitis in the vessels around; (d) Fusion or close approximation of the muscularis mucosæ and muscularis at the margin of the ulcer. All these points are usually, but not invariably, present in chronic peptic ulcers. *Table V* shows the number of gastric ulcers in which they were present in this series. It also shows their presence or absence in 88 chronic peptic ulcers in the duodenum or jejunum.

Usually in considering these criteria no attempt has been made to apply them to carcinomata in some other part of the alimentary tract where peptic ulcers do not occur. This has been done (Newcomb, 1930) with a series of 112 consecutive cases of carcinoma of the intestine removed surgically at St. Mary's Hospital between 1920 and 1930. Twelve cases had to be omitted as insufficient material was received for a complete examination. *Table V* includes the cases thought to be primary carcinomata and ulcer-cancer respectively.

Table V.—FREQUENCY OF FOUR CRITERIA IN THE HISTOLOGICAL DIAGNOSIS OF ULCER-CANCER.

MATERIAL	MUSCLE IN BASE OF ULCER		FIBROSIS			ARTERITIS		MUSCULARIS AND MUSCULARIS MUCOSÆ		
	Present	Absent	Slight	Mod.	Marked	Present	Absent	Separated	Unchanged	Fused
161 gastric ulcers..	5	156	—	—	161	116	45	—	2	159
Percentage of gastric ulcers	3	97	—	—	100	72	28	—	1·2	98·8
88 duodenal and jejunal ulcers ..	13	75	—	—	88	16	72	—	1	87
Percentage of 249 peptic ulcers ..	7	93	—	—	100	53	47	—	1·2	98·8
100 carcinomata of intestine ..	94	6	79	10	11	16	84	94	6	—
40 gastric carcinomata	38	2	22	2	16	6	34	40	—	—
Percentage of gastric carcinoma ..	95	5	55	5	40	15	85	100	—	—
6 ulcer-cancers ..	—	6	—	—	6	6	—	—	—	6

To consider these points in greater detail :—

a. From *Table V* it appears that the muscle is completely destroyed in the base of 97 per cent of all peptic ulcers. Of the five cases in which it remained, all were definite chronic ulcers with much fibrosis around. It is true that one has a history of only nine months, but the others have long histories, two of three years, one of twenty-five, and one of thirty-four. On the other hand, the muscle was completely destroyed in a considerable area of 6 per cent of carcinomata of the intestine where peptic ulceration is out of the question. It would seem then that this point, although a useful aid in diagnosis, is not absolute.

b. As by definition a chronic peptic ulcer is one in which periods of healing have taken place, it is obvious that fibrosis must be present in 100 per cent of cases. In the carcinoma of intestine group marked fibrosis was present in eleven cases. In one of them (*Fig.* 131) an area about 0·5 cm. in diameter

was free from cancer cells. In the others, although the growth was scirrhous in type, cancer cells were scattered in groups throughout the fibrous tissue. If a small area of cancer-free fibrosis can be formed in this way it seems possible that large areas may be also. As Paterson (1914) says, " We know that in cancer of the breast there may be formation of scar tissue, Nature's attempt at arrest of the disease."

It is noticeable in *Table V* that while only 11 per cent of the intestinal carcinomata show marked fibrosis, 40 per cent of the primary carcinomata of the stomach exhibit this point. It seems probable that this higher percentage of cases in the stomach may be due to secondary peptic ulceration of the cancer. It is probably easier for peptic ulceration to extend through a carcinomatous growth than through the normal stomach wall, for nearly all growths are more or less ulcerated. If, then, the normal tissues are reached by the peptic juice, a peptic ulcer may develop within or beneath the growth.

Fig. 131.—Carcinoma of large intestine. A. Carcinoma growing in submucosa and separating muscularis and muscularis mucosæ ; B, Area of dense fibrosis in centre of ulcer—groups of cancer cells are scattered in this part but some areas as large as a low-power field are cancer-free ; C. Muscularis completely destroyed between these points ; D, Vessels showing endarteritis. (× 3.)

The question then arises whether under these circumstances a good healing reaction will take place with the formation of dense fibrous tissue. That this does occur was suggested by Stromeyer (1912). It is fully discussed with the aid of most useful diagrams by Moskowicz (1924), his conclusion being that typical peptic ulcers often occur in or adjacent to carcinomata. A case in this series in which it is probable that this ulceration has occurred is described later.

A further possibility is that the peptic ulceration may be so rapid that the whole of the growth is destroyed. This has been put forward without much evidence by Morley Roberts (1926), who went so far as to suggest that every gastric ulcer is " a natural operation on a potential cancer." Duplant (1898) states that Tripier and Bard have seen cases of gastric ulcer whose cancerous nature was only demonstrable by the examination of metastases, the primary growth having been ulcerated away. He gives no further details or reference. Wilensky and Thalhimer (1918) described a case which both macroscopically and microscopically appeared a simple chronic peptic ulcer on the lesser curvature of the stomach. There was also a deposit of carcinoma in a neighbouring lymph-gland. They explain this as a case of maximum destruction of the growth with ulcer formation and the only evidence of the primary carcinomatous nature of the stomach lesion the metastatic growth in the gland. They give no clinical details, and a very weak spot in their argument is that the cancer was squamous-celled, a type which extremely rarely is primary in the stomach.

Several of the cancers of the intestine show the up-turning of the muscular coat into the floor of the lesion similar to that seen in chronic ulcer. One is illustrated in *Fig.* 132. It is quite clear in this case that it is due to contraction of the subserous fibrous tissue pulling the two halves of the ulcer together and so everting the edges of the muscularis. It has nothing to do with peptic ulceration.

c. Endarteritis or thrombophlebitis was present in 72 per cent of gastric ulcers. It is also present in 16 per cent of cancers of the intestine. It is, therefore, of little value as a diagnostic point in ulcer-cancer.

d. In the relationship between the muscularis mucosæ and muscularis we have a diagnostic point of great importance (*see Figs.* 131, 132). Fusion or close approximation of these layers was present in some part of 98·8 per

FIG. 132.—Carcinoma of jejunum. A rather scirrhous growth. The muscle is completely destroyed in centre of ulcer and its edges are upturned owing to the contraction of the fibrous tissue formed in the subserous region. The growth is spreading in the submucosa separating the muscularis mucosæ and muscularis. (× 3.)

cent of the peptic ulcers in this series. The two cases where it was absent were in the stage of rapid extension all round the ulcer, a very rare condition. In all the other gastric ulcers definite signs of healing were taking place with fusion or approximation of the muscularis mucosæ and muscularis. Stewart (1928 and 1929), while agreeing that if fusion be " present in association with carcinoma assuredly the latter is secondary even although there may be considerable infiltration of the floor ", objects that fusion is an indication of healing rather than of chronicity. But what is fibrosis ? Surely fibrosis on which he rightly lays great stress is also an indication of healing rather than chronicity. He further states that he has numerous specimens of uncomplicated chronic ulcer in which the muscularis mucosæ and muscular coat are widely separated. In this series no case occurred where they were separated widely.

In the intestinal carcinoma group the muscularis mucosæ and muscularis were widely separated in 94 per cent, unchanged in 6, and in no case were they approximated or fused. It would appear, then, that fusion is the only one of these four points which is absent in all the cases of carcinoma of the intestine ; it is present in all the cases of healing peptic ulcers, and it is therefore suggested that it is the most important point in the histological diagnosis of the pre-existence of peptic ulcer.

2. **Evidence of Cancer**.—The diagnosis of malignancy of a tumour in the stomach is no different from its diagnosis in other organs. The best evidence is the presence of metastatic growth in glands or elsewhere. This is definite proof of malignancy when present, but in most cases will not have occurred. It is, therefore, not of much practical value.

Usually it is necessary to fall back on the more debatable grounds of variation in appearance and arrangement of individual cells and local

infiltration. The following points should be considered. Atypical arrangement of cells in solid masses or columns instead of in tubes or acini ; variation in size and shape, especially the formation of polygonal or polyhedral instead of cubical or columnar cells ; variation in nuclei, including their central position and hyperchromatism ; a large number of mitotic figures, especially if they are abnormal. All are points in favour of malignancy, but any may be present in rapidly regenerating epithelium. Many of them taken together would strongly suggest malignancy and necessitate a careful examination of other sections for

FIG. 133.—Edge of chronic gastric ulcer in healing stage. (× 3.)

further evidence.
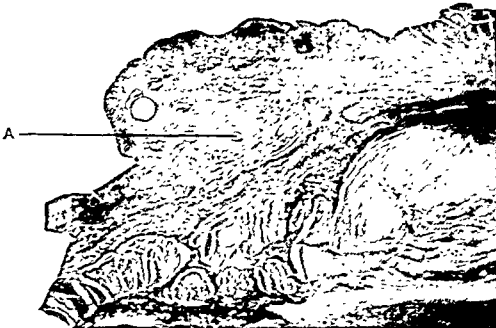
FIG. 134.—Edge of chronic gastric ulcer in healing stage, showing heterotopic epithelium (A) in dense fibrous tissue. (× 10.)

Infiltration of other tissues by epithelium is usually accepted as evidence of malignancy, but it is necessary to be certain that the epithelium is growing into the tissue and not merely misplaced as the result of congenital defect or from irregular healing. Heterotopic epithelium is not carcinoma. It is usually arranged in definite tubes or acini. The cells are regular with basal nuclei. In this connection care must be taken not to confuse oblique cutting of a tube with heaping up of epithelium. *Figs.* 133–135 illustrate this point well.

It is probable that some of the high figures given for the proportion of cancer arising in ulcer are caused by mistaking this heterotopic epithelium for cancer. In this series it was present in 44 of 161 ulcers, or nearly 28 per cent. Dible (1925) found it in 32 per cent of his series, and Taylor (1927) in 22 per cent. If it had been taken as evidence of carcinoma in this series, it would have brought the number of ulcer-cancers up to 50, and the percentage of carcinoma arising in ulcer would be 55·5, and that of ulcers becoming malignant 31.

To summarize : for the histological diagnosis of ulcer-cancer, there must

FIG. 135.—The alveolus marked A in *Fig.* 134 under higher magnification. The epithelium appears to be several layers thick, but serial sections showed that this was owing to oblique cutting of edge of alveolus. (× 100.)

be definite evidence of both carcinoma and of pre-existing peptic ulcer. The only definite evidence of the latter not given occasionally by primary carcinomata is fusion of the muscularis mucosæ and muscularis at the edge of the ulcer. It is suggested that the presence of this criterion is as valuable as the demonstration of tubercle bacilli in the diagnosis of tuberculosis.

APPLICATION OF CRITERIA TO THIS SERIES.

Applying the criteria to this series there are only six cases which are definitely carcinoma arising in pre-existing peptic ulcer. They will be considered in detail.

Case 1 (*Figs.* 136, 137).—Male, age 33, admitted with 5 years' history of pain half an hour after food, relieved by vomiting. He was diagnosed clinically as gastric ulcer. At operation an ulcer on the lesser curvature near the pylorus was found
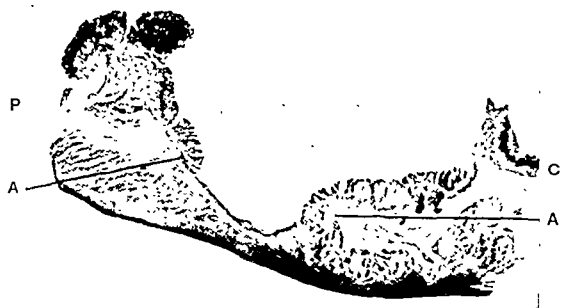
FIG. 136.—*Case* 1. Ulcer-cancer of stomach. C and P mark cardiac and pyloric sides respectively. Small nodules of growth can be seen infiltrating the muscularis on the cardiac side. Fusion of muscularis mucosæ and muscularis is seen at A. (× 3·6.)

and carcinoma was not suspected until the microscopic sections were examined. Macroscopically the ulcer was 1·2 cm. in diameter, had hard raised edges, the lower one involving the pyloric ring. Microscopically the section shows a typical chronic

FIG. 137.—*Case* 1. The cardiac side of specimen shown in *Fig.* 136 with higher magnification. Fusion of muscularis mucosæ and muscularis is seen at A. Infiltration of the muscle by the cancer is shown just below this. (× 12.)
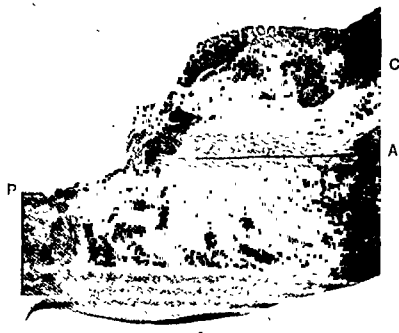
peptic ulcer. The muscularis is completely destroyed in its base, where there is a large fibrous mass. Fusion of muscularis mucosæ and muscularis is shown at both edges, and several vessels with endarteritis are present. On the cardiac side of the ulcer there is a mucous carcinoma which is definitely invading the muscularis.

Case 2 (*Figs.* 138–141).—Male, age 53. History of eight to nine years' attacks of 'catarrh of stomach'; six months' flatulence and feeling of fullness; seven

weeks' frequent vomiting. Loss of weight; no appetite. At operation an ulcer was found on the lesser curvature towards pyloric end. Wedge resection performed. Macroscopically a wedge-shaped portion of stomach involving 7 cm. of lesser curvature. An ulcer 2 cm. in diameter with raised edges occupies its centre. Half the specimen is seen in the coloured drawing (*Fig.* 138). Microscopically, an ulcer with carcinoma in its edges and base. In one section only there was complete destruction of the muscle. There is much fibrosis around and in the base, but it is infiltrated with carcinoma of an undifferentiated polygonal-celled type. Many vessels showing endarteritis are present and fusion of muscularis mucosæ
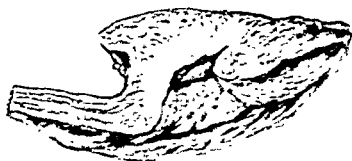
Fig. 138.—*Case* 2. One half of an ulcer-cancer of the stomach. The cut surface is close beside and parallel to the lesser curvature. (× ⅔.)

and muscularis at the edges. Owing to this last fact it is placed in the ulcer-cancer group in spite of the presence of muscle in the base of the ulcer in most sections and the infiltration of the base with carcinoma.
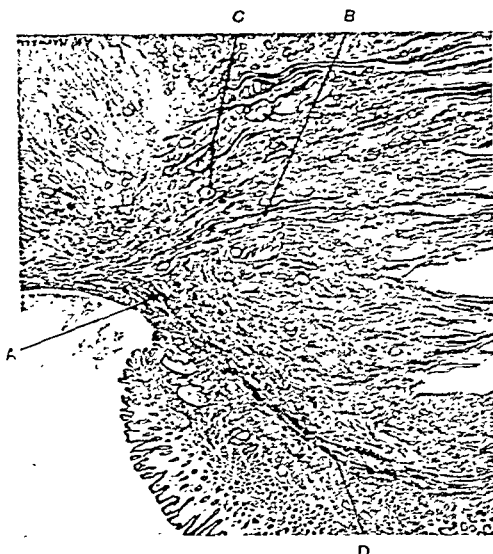
Fig. 139.—*Case* 2.—Microphotograph (retouched) of edge of ulcer. At A the fusion between muscularis (B) and muscularis mucosæ (D) can be seen. C, Acini of carcinoma invading muscle. (✗ 24.)

Fig. 140.—*Case* 2. Edge of ulcer-cancer showing fusion of muscularis mucosæ and muscularis, and invasion of muscle by the growth. (× 12.)
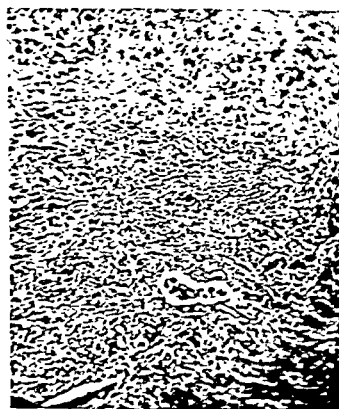
Fig. 141.—*Case* 2. A portion of *Fig.* 140 under higher magnification to show a group of cancer cells in a lymphatic. (✗ 100.)

Case 3 (*Figs.* 142, 143).—Male, age 53. History of three years' attacks of pain in epigastrium soon after food; one year vomiting after evening meal. The

pain, which has no relation to food. No vomiting. At operation chronic ulcer of lesser curvature adherent to pancreas. Macroscopically, pylorus and portion of stomach with ulcer 2 cm. in diameter on lesser curvature. Cardiac side overhanging. Microscopically, a chronic peptic ulcer, recently active. The muscle is completely destroyed and replaced by dense fibrous tissue. Endarteritis present in several vessels. Fusion of muscularis mucosæ and muscularis. On the overhanging cardiac side there is much atypical epithelial proliferation. The cells vary in size and shape. Many are polygonal and arranged in solid trabeculæ. Hyperchromatic nuclei and mitotic figures present. The first sections examined showed no definite signs of carcinoma; further blocks were cut and infiltration of lymphatics demonstrated. This is the only case where the diagnosis of carcinoma presented any difficulty.

DURATION OF SYMPTOMS.

Stewart (1929), Dible (1925), and others lay considerable stress on the value of the clinical history in arriving at a diagnosis of ulcer-cancer. They rightly point out that a patient who is operated on for gastric ulcer has usually suffered from indigestion for a considerable number of years, while in their experience most cancer patients have a short history. They both go so far as to exclude certain cases from their ulcer-cancer group owing to the short history. This would seem to indicate a lack of faith in their histological criteria.

The possible fallacy in stressing the clinical history is well shown in one case. A man, age 50, commenced his illness by having hæmatemesis in 1920. After this he suffered from attacks of abdominal pain. In 1923 he was admitted to hospital and a laparotomy was performed. The stomach and duodenum were normal and his appendix was removed. As the pain recurred he was again operated on in 1925, and a typical chronic peptic ulcer on the lesser curvature was excised. Did his symptoms date from 1920 or 1923 ?

In obtaining clinical histories for the series of cases here described there has been considerable difficulty. In most instances the notes in the hospital records were written by junior dressers and points of great importance were omitted. One of the worst difficulties was the habit of starting the history with a previous operation. In several cases it was impossible to find out why the first operation was performed and the length of illness prior to it. In a few cases I have personally seen patients with short histories and have been told, " of course I have had indigestion all my life," when the dresser had recorded " three months' pain after food " and " previous history good ". However, the best histories available have been obtained and are shown in Table VI.

Table VI.—DURATION OF SYMPTOMS IN MONTHS AT TIME OF OPERATION.

	ULCER	CANCER	CANCER CORRECTED	ULCER-CANCER
Shortest ..	2	1	1	36
Average ..	112	39	9	71
Longest ..	480	540	48	114

In the carcinoma group there are four patients who all dated their illnesses for comparatively short times, but who have recorded under 'previous illnesses' a long period of indigestion, in one case lasting for forty-five years. Two sets of figures, therefore, are given in the table, the first showing the duration from the onset of indigestion and the second from the commencement of the symptoms for which the patient was admitted to hospital.

Counting the four cases mentioned above, nine of those included in the primary carcinoma group have histories lasting for three years or over. The other thirty-one all dated the onset of their symptoms within two years of operation. Of the nine cases eight presented none of the four histological criteria of pre-existing peptic ulcer mentioned above and are definitely ulcerated cancers. The ninth is a case with considerable difficulty and is described in full.

Case 7.—A man, age 47, came to hospital for hæmatemesis and melæna. He had a history of two years' pain and vomiting after food. After a few weeks' rest he was radiographed and an ulcer with a double crater and thick edges was seen on the lesser curvature. It was reported ·strongly suggestive of carcinoma'. As, however, the pain and vomiting had now disappeared he refused a laparotomy and went home. He was readmitted ten months later with a return of symptoms. A second radiogram showed very little change in the ten months. Laparotomy was performed and a large piece of stomach removed. On examination a double ulcer 4·2 cm. × 3·5 cm. was found on the lesser curvature as described by the radiologist, and the raised edges strongly suggested carcinoma (*Fig.* 146). Microscopically, the pyloric crater is a typical ulcerated carcinoma. The base contains muscle with scirrhous growth running between its fibres. The growth is spreading in the submucosa and separating the muscularis mucosæ from the muscularis (*Fig.* 147). In the adjoining crater the muscle is completely destroyed and replaced by fibrous tissue and growth. An area 0·3 cm. wide of fibrous tissue is free from growth. At the edges the muscularis mucosæ and muscularis are again separated. Several small vessels showing endarteritis obliterans are present.
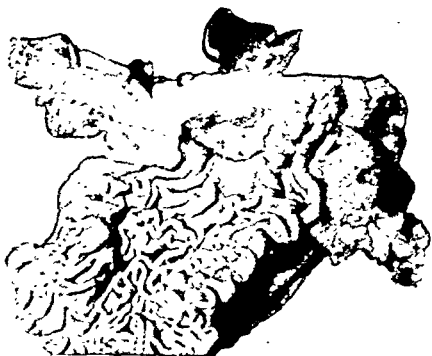
FIG. 146.— *Case* 7. Ulcerated carcinoma of stomach. The anterior half of piece of stomach removed at operation. A double crater is shown on the lesser curvature and there is complete destruction of the muscle by the cardiac crater. (× ¼.)

FIG. 147.—*Case* 7. Section of the same case show ·ing pyloric crater to be a typical ulcerated carcinoma with peptic ulceration on its cardiac edge. The muscularis mucosæ is separated from the muscularis. (*Natural size.*)

Here, then, is a case where all the criteria for the diagnosis of ulcer-cancer are present except one—the fusion of muscularis mucosæ and muscularis. It is suggested that the growth started near the pylorus and that there was

later peptic ulceration on the cardiac side of it, destroying it and so allowing peptic digestion of the muscle in the region of the cardiac crater to take place. Fibrous tissue has then formed in the submucous region and by contracting has given the upturned appearance of the muscle. It seems that the histological appearance can be explained on the assumption that it was a primary carcinoma with secondary peptic ulceration.

Another interesting feature of the case is that it was possible by X rays to see the double ulcer ten months earlier in a condition similar to that in which it was found at operation. This suggests a very slow rate of growth for the carcinoma and so gives an explanation of the comparatively long history.

Paterson (1914) calls attention to two of his cases with very long histories. One remained well for three years after gastrojejunostomy for inoperable carcinoma. The second was alive four and a half years after a similar operation. He suggests that the natural history of gastric carcinoma is very variable and may be long. It is wrong to assume that a case of gastric carcinoma arose in an ulcer merely because it has a long history.

AGE AND SEX.

Age.—*Tables VII* and *VIII* show the age at operation and at onset of symptoms in the three groups. The numbers in the carcinoma and ulcer-cancer groups are obviously much too small to warrant any conclusions, but they agree rather closely with Professor Stewart's and show that ulcer-cancer is intermediate in age between ulcer and cancer.

Table VII.—AGE IN YEARS AT OPERATION.

	ULCER	ULCER-CANCER	CARCINOMA
Youngest	22	30	33
Average	44·6	45	45
Stewart's average	44	48	50
Oldest	66	56	72

Table VIII.—AGE IN YEARS AT ONSET OF SYMPTOMS.

	ULCER	ULCER-CANCER	CARCINOMA	CORRECTED CARCINOMA
Youngest	16	28	16	33
Average	35	40	52	54
Stewart's average	36½	41½	49	—
Oldest	63	53	71	71

Sex.—In the six cases of ulcer-cancer here recorded 4 were males and 2 females. Stewart records 9 males to 2 females. Cabot and Adie's 5 cases were all males. Dible's were male and female. Adding these together: of

24 cases, 19 were males and 5 females. The number of cases is obviously too small to allow of any definite conclusions, but they do suggest a greater frequency of ulcer-cancer in the male.

FREQUENCY OF ULCER-CANCER.

Estimates of the frequency of ulcer-cancer can be made in two ways: the proportion of cases of carcinoma of the stomach which arise in peptic ulcers, or the proportion of cases of peptic ulcer which become carcinomatous. The conclusions of 102 observers of the first percentage are shown in *Fig.* 117 (p. 281) and in the Appendix (p. 305). The extreme variations are probably due to lack of definite criteria on which to base the diagnosis of ulcer-cancer. Some of the figures are based on clinical examinations, some on radiological, some on histories, only comparatively few on exact morbid anatomical studies. Even with the most definite histological criteria the figures obtained can only apply to a selected group of cases, either those coming to operation or those coming to post-mortem. These considerations apply even more strongly to estimates of the second kind—the percentage of cases of peptic ulcer becoming carcinomatous.

The figures in this series are shown in *Table IX*, Stewart's figures again being given for comparsion.

Table IX.—Frequency of Ulcer-Cancer.

				Stewart
Total cases of peptic ulcer	..	..	.. 160	180
Total cases of carcinoma	..	..	.. 46	70
Total cases of ulcer-cancer	..	..	.. 6	11
Percentage of ulcers becoming cancer ..		..	3·75	6·1
Percentage of carcinoma arising in ulcer		..	13	15·7

It is important to keep clearly in mind the significance of these figures. They cannot be applied to the general population but only refer to a very limited group—namely, those whose ulcers have been excised. This group only includes a fraction of the cases admitted to hospital and takes no notice of those treated medically or by gastrojejunostomy. During the period 1920 to April, 1931, there were admitted to St. Mary's Hospital 731 cases of gastric ulcer, of which only 160, or rather more than one-fifth, are considered in this series: 339 cases of gastric carcinoma were admitted in the same period, and in only 46 (about one-seventh) was surgical removal performed. Again, in each clinic where similar estimations are made, the figures will depend on the method of selection of cases for operation, and whether excision is the routine treatment. Orator (1925) estimated in his series that 30 per cent of ulcers in the prepyloric region had become cancerous, while only 2 per cent in the rest of the stomach had become so changed. Finsterer (1925) pointed out that a higher proportion of cases of cancer is present among hard callous ulcers than among softer ones. Some surgeons only remove callous ulcers, and others only those near the pylorus. Both of them will have a higher percentage of ulcer-cancer than those who remove nearly all ulcers operated upon. The majority of the cases considered in this paper come from one surgeon whose routine treatment of gastric ulcers is excision, and consequently the percentage of ulcer-cancers is comparatively low.

Hauser (1926) has collected statistics from several German workers who have published estimates of the frequency of ulcer-cancer based upon morbid anatomical considerations.   It is reproduced here (*Table X*).

Table X.—FREQUENCY OF ULCER-CANCER.

OBSERVER		ULCERS	ULCER-CANCERS	PERCENTAGE
Scheuerman (München)	..	96	1	1
Berthold (Berlin) ..	..	294 ·	4	1·3
Wolowelsky (Basel) ..	..	139	2	1·4
Cohn (Kiel)	..	295	6	2·0
Greiss (Kiel) ..	..	136	4	2·9
Schneider (München) ..	..	89	3	3·4
Brinkman (Kiel) ..	..	725	40	5·5
Total ..	..	1774	60	3·4 (Average 2)

These figures agree with most of the rather conservative estimates made by other morbid anatomists such as Dible (1925), who found 1·7 per cent in 128 ulcers, and Kummer (1919) with 2 per cent in 357 ulcers.   Ewing (1918) gives a good historical review and critical examination of the various criteria proposed by numerous authors.   He gives no figures of his own, but concludes that the cancerous change does not take place in more than 5 per cent of ulcers.

Very different figures have been given by the pathologists of the Mayo Clinic, who consider that the majority of ulcers removed surgically are undergoing malignant change.   These views will be considered critically later.

Attempts to estimate the frequency of ulcer-cancer have been made by tracing the after-history of cases treated by gastrojejunostomy or after perforation of peptic ulcers.   Paterson (1914) estimated that only 1 per cent subsequently developed carcinoma.   Greenough and Joslin (1899) found 1 in 112 cases, and Bamberger (1909) collected 22 in 1025 gastrojejunostomies or 2·1 per cent.   These figures, however, do not prove that the carcinoma developed in the pre-existing ulcer, and cannot be accepted without reservation. Owen (1926) described the post-mortem on a man who died ten years after a gastrojejunostomy.   He found an annular carcinoma of the pyloric antrum reaching up to but not including the scar of a healed peptic ulcer on the lesser curvature, but there was no evidence that the growth started in the ulcer. I have seen a similar case at post-mortem five years after a gastrojejunostomy. In this case the scar and growth were quite distinct.

In the series described here one case showed both an early carcinoma near the pylorus and a typical chronic peptic ulcer on the lesser curvature. They were widely separated by normal stomach mucosa, and no evidence exists that they had any connection with each other.   It is quite clear, then, that proof of carcinoma occurring in a stomach known to have contained an ulcer is not proof that the carcinoma originated in the ulcer.

CRITICAL EXAMINATION OF LITERATURE.

In reading the voluminous literature on this subject two points are at once noticeable. First the large number of obvious errors, arithmetical and otherwise. In many cases percentages are incorrectly calculated, while in others charts do not correspond with the figures given in the text. Cabot and Adie (1925), in their otherwise excellent paper, make both these mistakes. They refer to 50·6 per cent of 82 observers, which presumably means 41½. Their chart referring to the same 82 observations is drawn in a similar way to *Fig.* 117. Only one observer estimates a percentage of 91 to 100, and so the unit can be measured. Applying this unit to the other parts of their curve, the total number of observers works out at 133 instead of 82. Another common error is misquotation of previous papers and incorrect references. This was a source of considerable annoyance in the compilation of the chart in *Fig.* 117. Secondly, in very few papers is it possible to find the author's criteria of ulcer-cancer. In some the diagnosis appears to rest on the history, in others it is radiological, while in many papers those diagnosed histologically are not separated from others diagnosed only on the macroscopic structure.

It is obviously only possible to consider a few of the more important papers in detail.

MacCarty, of the Mayo Clinic, has published a long series of papers on this subject. In 1909, with Wilson, in the *American Journal of the Medical Sciences*, he enumerated 218 cases :—

	Cases
Duodenal ulcer	8
Simple gastric ulcer ..	47
Carcinoma on ulcer	109
Some evidence of pre-existing ulcer	11
No evidence of ulcer..	33
Sarcoma	2
Adenoma ..	2
Diverticulum	1
Possible transition cases	5
Total	218

Then in 1914 he publishes another paper with Broders, based on 684 cases, including the 218 previously reported. These he classifies :—

	Cases
Simple ulcers	191
Ulcer + carcinoma ..	472
Ulcer + doubtful carcinoma ..	21
Total	684

In the five years between 1909 and 1914, 2 sarcomas, 2 adenomas, a diverticulum, and 33 cases with no evidence of ulcer have been reclassified. He gives no explanation at all of this. He gives the protocols of 12 cases of carcinoma in ulcer. One case from the photograph of the macroscopic specimen might be an ulcer-cancer, but the others look more like ulcerated primary cancer. The microphotographs are too poorly reproduced to criticize. In some cases the histories are used for diagnosis, in others they are completely discounted.

In April, 1927, he complains of misquotation and false understanding and quotes the same figures from his previous papers, again making no attempt

to explain the discrepancies referred to above. He states that his diagnosis is now based largely upon frozen sections of unfixed tissues, and that this method is more reliable than fixed paraffin sections.

It is impossible to answer this last statement without a much more extensive experience of frozen sections of unfixed tissues than I possess, but in this respect a paper from Rochester, N.Y., by W. J. Scott (1928), is of interest. He describes an ulcer on the lesser curvature associated with a nodule on the duodenum. With fresh frozen sections the pathologist reported the ulcer benign and the duodenal nodule malignant. Later paraffin sections of the fixed tissues showed the ulcer malignant and the duodenal nodule to be ectopic pancreatic tissue. Such a report from the other Rochester gives rise to a feeling of doubt as to the trustworthiness of MacCarty's observations.

C. D. Aaron (1921) writes that Albert Kocher states that he has personally examined the Mayos' specimens and is convinced that much of what they labelled cancerous degeneration of ulcers was in reality merely atypical proliferation of epithelium. He gives the reference—" Chronic and Duodenal Ulcer, *Correspondenz-Blatt für schweizer Aerzte*, Basel, May 10th, 1919, No. 19 ", which is incorrect. The correct reference, if any, has not been found, and consequently the statement cannot be confirmed, but it seems the most likely explanation of the high figures coming from the Mayo Clinic.
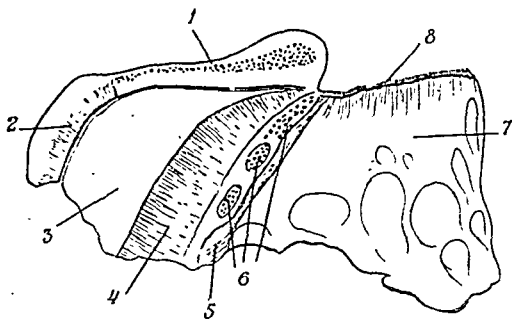
Fig. 148.—Copy of *Fig.* 123 from Pauchet and Hirchberg's book, *Cancer d'Estomac*, showing fusion of muscularis mucosæ and muscularis at edge of ulcer.

Of the papers where histological details are given, Dible's (1925) is one of the most complete. There seems little doubt that the two cases he accepts are definite ulcer-cancers. He also described three other cases which he considered might be ulcer-cancers from their histological appearance, but discarded them owing to their short clinical histories. As far as can be seen from his pictures, they would not fit in with my criteria, but it is remarkable to find a pathologist who has more faith in clinical histories than his own microscopical findings.

The importance of fusion of the muscularis mucosæ and muscularis in the histological diagnosis of ulcer-cancer was first brought forward by Professor H. M. Turnbull in his lectures at the London Hospital. It was demonstrated at the Royal Society of Medicine and briefly reported in the *Lancet* (Newcomb,

1925), and Stewart (1929) discusses it. No one else appears to have recognized its importance, although it can be seen in one of Dible's illustrations and in two cases described by Hirchberg (1928). A photograph of a diagram from his book is reproduced in *Fig.* 148. It is curious that in spite of illustrating it, he does not mention it in the description.

It had been hoped to apply the criterion of fusion of muscularis mucosæ and muscularis to the cases described by other workers on this subject, but in no paper were sufficient histological details given for this to be done.

SUMMARY.

1. The wide diversity of opinion with regard to the etiological relationship between peptic ulceration and gastric carcinoma has been pointed out.

2. It is suggested that this has arisen from a lack of definite histological criteria for the diagnosis of ulcer-cancer.

3. Four histological criteria for the diagnosis of ulcer-cancer have been considered and applied to control groups of 249 peptic ulcers and 100 carcinomata of intestine.

4. The great importance of fusion between the muscularis mucosæ and muscularis at the edge of the ulcer in this diagnosis has been pointed out.

5. Application of this test to a series of cases shows that 3·75 per cent of ulcers showed malignant change and 13 per cent of the cancers showed evidence of previous peptic ulceration.

My grateful thanks are due to my old chief, Professor H. M. Turnbull, for his teaching, without which this paper would never have materialized, and also to the Surgical Staff of St. Mary's Hospital, and in particular Professor C. A. Pannett, for providing the material on which it is based.

APPENDIX.

List of percentages of cases of carcinoma of stomach estimated by various authors as having arisen in pre-existing peptic ulcer. The figures in brackets after the percentage represent the total number of cases studied by that author.

YEAR.	PER CENT.	OBSERVER AND REFERENCE.
1890	0	Kollmer (*Berl. klin. Woch.*, 1890, v, 119 and 146).
1897	0 (59)	Krokiewicz (*Wien. klin. Woch.*, 1897, 1127).
1898	0	Duplant (*Thèse de Lyon*, 1898, No. 132).
1904	0 (66)	Peterson and Colmers (*Beitr. z. klin. Chir.*, 1904, xliii, 1).
1904	0	Pförringer (*Wachstum des Magencarcinoms, Ibid.*, 1904, xli).
1906	0	R. Schmidt (*Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1906, xv, 701).
1914	0	Paterson (*Surgery of the Stomach*, 2nd ed., 1914, 276).
1922	0	F. Peyser (*Deut. Zeits. f. Chir.*, 1922, clxviii, 404).
1923	0 (50)	Duggan (*Boston Med. and Surg. Jour.*, 1923, clxxxix, 471).
1928	0	Izod Bennett (*Brit. Med. Jour.*, 1928, i, 170).
1901	1 (361)	Borrmann (*Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1901, 2).
1848	1·2 (160)	Dittrich (*Vierteljahr. f. d. prakt. Heilkunde*, 1848, xvii, 1).
1913	1·2	Gruber (*Zeits. f. Krebsforsch.*, 1913, xiii).
1890	2	Eichhorst (*Handb. d. spez. Pathol.*, 4 Aufl. Bd. 2, 136).
1904	2·2	Cooper and Shaw (*Guy's Hosp. Rep.*, 1904, xliii, 121).
1901	2·6	Osler (Osler and McCrae, *Principles and Practice of Medicine*, 10th ed., 495).

YEAR.	PER CENT.	OBSERVER AND REFERENCE.
1909	2·3	Bamberger (*Die Innere und die chir. Behand. des chr. Magengeschwürs.* Berlin).
1918	2 (48)	Wilensky and Thalhimer (*Ann. of Surg.*, 1918, lxvii, 215).
1929	2·7	Pettinari, V. (*Tumori*, 1929, xv, 248, etc.).
1859	3	Plange (Dissert. Inaug., Berlin, 1859, quoted by Rosenheim).
1883	3	Berthold (Dissert. Inaug., Berlin, 1883, quoted by Rosenheim).
1889	3	H. Häberlin (*Deut. Arch. f. klin. Med.*, 1889, xliv, 461).
1902	3	Fenwick (*Cancer and other Tumours of Stomach*, 102).
1911	3–7	Lockwood (*Jour. Amer. Med. Assoc.*, 1911, lvi, 951).
1913	3–5	Konjetzny (*Beitr. z. klin. Chir.*, 1913, lxxxv, 455).
1920	3–5	Anschütz (*Deut. Zeits. f. Chir.*, 1920, cliv, 1).
1928	3·5	De Vecchi (*Arch. ital. di Chir.*, 1928, xxxi, 1).
	3·3 (331)	Herschenson (quoted by Pettinari).
	3–5	Winternitz (quoted by Poscharissky).
1868	4	Steiner (Dissert. Inaug., Berlin, 1868, quoted by Rosenheim).
1868	4	Wollmann (Dissert. Inaug., Berlin, 1868, quoted by Rosenheim).
1902	5 (228)	Riechelmann (*Berl. klin. Woch.*, 1902, xxxix, 728).
1907	5 (176)	Redlich (*Zeits. f. Krebsforsch.*, 1907, v, 261).
1915	5	Max Einhorn (*Jour. Amer. Med. Assoc.*, 1915, lxv, 1230).
1927	5	Stanley Wyard (*Diseases of the Stomach*, 329).
1902	5·6 (159)	Borst (*Lehr. von den Geschwülsten*, Wiesbaden, 1902, quoted by Pettinari).
1882	6·1	Danielsen (Inaug. Diss., Würzburg, 1882, quoted by Poscharissky).
1901	6 (241)	Borrmann (*Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1901, 2).
1912	6·8	Perthes (*Münch. med. Woch.*, 1912, lix, 2140).
	6·8	Altschul (quoted from Pettinari).
1925	6	Dible (*Brit. Jour. Surg.*, 1925, xii, 666).
1928	6	Bastianelli (*Report of International Conference on Cancer*, 1928, 355. London).
1913	7 (60)	Saltzmann (*Studien über Magenkrebs aus dem Univ. Helsingfors*, 1913. Jena).
1914	7·3 (1000)	Friedenwald (*Amer. Jour. Med. Sci.*, 1914, cxlviii, 660).
	7	Casolo (quoted by Pettinari).
1890	8 (50)	Rosenheim (*Zeits. f. klin. Med.*, 1890, xvii, 116).
	8·2	Menne (quoted by Poscharissky).
	8·5	Petren (quoted by Anschutz and Konjetzny).
1878	9	Lebert (*Krankheiten des Magens*, 1878, 440. Tübingen).
1925	9	Cabot and Adie (*Ann. of Surg.*, 1925, lxxxii, 86).
1926	10	J. S. Lawrence and Boch (*Boston Med. and Surg. Jour.*, 1926, cxcv, 651).
1882	10–15	Litten (*Berl. klin. Woch.*, 1882, xix, 658).
1925	10–15	Orator (*Virchow's Arch.*, 1925, cclvi, 202).
	11·4	Eppinger (*Prager Vierteljahr*, cxiv, quoted by Welch).
1917	12·2 (43)	Fichera (*Tumori*, 1917, quoted by Pettinari).
1890	13 (38)	Hauser (*Das Cylinderepithel-carcinom des Magens*, 1890. Jena).
1912	13·3 (230)	Genzken (Inaug. Diss., Kiel, 1912, quoted by Konjetzny).
1892	14 (156)	Sönnichsen (*Zeits. f. klin. Med.*, 1892, xvii, 117).
1900	14·7 (163)	Tiemann (Inaug. Diss, Kiel, 1900, quoted by Konjetzny).
1914	15	Küttner (*Lang. Arch. f. klin. Chir.*, 1914, cv, 788).
1929	15·7	Stewart (Hurst and Stewart, *Gastric and Duodenal Ulcer*, 1929, 381).
1930	15	Poscharissky (*Zeits. f. Krebsforsch.*, 1930, xxxi, 263).
	15·6	Woloschin (quoted by Poscharissky).
1909	16 (662)	Haberfeld (*Zeits. f. Krebsforsch.*, 1909, vii, 190).
1910	16·5	Matti (*Deut. Zeits. f. Chir.*, 1910, civ, 425).
1895	17	Buttenberg (Inaug. Diss., Jena, quoted by Audistère).
1921	17	Taylor and Miller (*Amer. Jour. Med. Sci.*, 1921, clxii, 862).
1928	17·3 (98)	Duval and Moutier (*Bull. et Mém. Soc. nat. de Chir.*, 1928, liv, 423).
	18	Reimer (quoted by Audistère).
1896	19 (42)	Hemmerschlag (*Arch. f. Verdauungskrank.*, 1896, ii, 1).
	19	Miroljuboff (quoted by Poscharissky).
1931	(less than 20)	Walton (*Lancet*, 1931, i, 1072).
1929	20	Hurst (*Lancet*, 1929, ii, 1023).
	20	Rivier Jaboulay (quoted by Rodman).
1928	22 (94)	Hayem (*Presse méd.*, 1928, xxxvi, 1137).

YEAR.	PER CENT.	OBSERVER AND REFERENCE.
1900	26·7 (120)	Klausa (Inaug. Diss., Kiel, 1900, quoted by Hauser).
1904	26	Mazdl-Jedlicka (*Zur operat. Behand. des chron. Magengeschwürs*, 1904. Prague).
1922	26	Payr (*Arch. f. klin. Chir.*, 1910. xciii, 436).
1903	30	Stich (*Bruns. Beitr. z. klin. Chir.*, 1903, xl, 342).
1909	30	Riedel (*Deut. med. Woch.*, 1909, quoted by Payr).
1930	30	Finsterer (*Arch. f. klin. Chir.*, 1930, clix, 30).
1925	37	Finsterer (*Wien. klin. Woch.*, 1925, xxxviii, 295).
1928	37 (35)	Spriggs (*Report of International Conference on Cancer*, 1928, 335. London).
1919	over 40	C. H. Mayo (*Ann. of Surg.*, 1919, lxx, 236).
1923	41	Sherren (Choyce, *System of Surgery*, 2nd ed., ii, 393).
1908	50	Rodman (*Jour. Amer. Med. Assoc.*, 1908, 1, 165).
1919	50	Deaver (*N.Y. Med. Jour.*, 1919, cix, 749).
1915	above 50	W. F. Cheney (*Jour. Amer. Med. Assoc.*, 1915, lxv, 1227).
1914	52–57	Wilson and McDowell (*Amer. Jour. Med. Sci.*, 1914, cxlviii, 796).
1906	54	Graham (*Boston Med. and Surg. Jour.*, 1906, clv, 193).
1907	54 (69)	W. J. Mayo (*Ann. of Surg.*, 1907, xlv, 810).
1907	59·3	Mayo Robson (*Cancer of Stomach*, Bradshaw Lecture).
	62	Paterson (quoted by Poscharissky).
1909	66·6	Moynihan (*Brit. Med. Jour.*, 1909, i, 830).
1917	66	Smithies (*Illinois Med. Jour.*, 1917, xxi, 149).
1910	67	Wilson and Willis (*Collected Papers of Mayo Clinic*, 1910, 118).
1914	68·6 (493)	McCarty and Broders (*Arch. of Internal Med.*, 1914, xiii, 208).
1909	71 (153)	Wilson and McCarty (*Amer. Jour. Med. Sci.*, 1909, cxxxviii, 847).
1920	75	Pauchet and Delort (*Presse méd.*, 1920, ii, 793).
1905	82 (50)	Mumford and Stone (*Surgical Aspects of Digestive Disorders*, 1905. New York).
1910	over 80	Mansell Moullin (*Lancet*, 1910, i, 415).
1900	over 90	Ssapeshko (*Annalen d. Russ. Chir.*, 1900, No. 6, quoted by Rodman).

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A REPORT ON THE STRANGEWAYS COLLECTION OF RHEUMATOID JOINTS IN THE MUSEUM OF THE ROYAL COLLEGE OF SURGEONS.

By R. LAWFORD KNAGGS,

With Photo-micrographs by G. H. RODMAN.

PART II.

SOME NATURAL FEATURES AND HISTOLOGICAL PECULIARITIES OF OSTEO-ARTHRITIS AND RHEUMATOID ARTHRITIS.

I. OSTEO-ARTHRITIS.

A. The Histology of Osteophytes.

1. **The Development of an Osteophyte.**—The present view of the origin of an osteophyte is that it is due to excitation of the adjacent medulla. Vascular cellulo-connective tissue, supposed to be an extension from the medulla,* appears in patches of softening, or in spaces arising from them, in the deep parts of the articular cartilage. The cartilage becomes honeycombed with these spaces, and the intervening cartilage is converted into bone by metaplasia.

There is no question that in an osteo-arthritic joint spaces filled with vascular cellulo-connective tissue appear in the deep layers of the articular cartilage, but the contents arise from proliferation of the connective-tissue basis of the cartilage and a reversion of the cartilage cells to the connective-tissue type. After the formation of these spaces the evolution of the osteophyte begins.

a. The cartilage bordering the spaces may undergo more or less calcification, and bone is developed in the cartilage or in the calcified parts of it by metaplasia.

b. The adjacent cartilage loses its cartilaginous appearance, changing into fibrocartilage or fibro-connective tissue, and additional spaces are outlined in it as calcification and ossification advance from the periphery of the developing osteophyte.

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* There is but little justification for this idea, for the adjacent medullary spaces are in some cases filled with fat only, and in others besides the fat there may be only a small development of proliferated connective tissue. Moreover, in some instances where the section is successful in demonstrating an aperture in the subchondral bone lamina connecting a medullary space with the spaces of a forming osteophyte, the change from fat medulla to the proliferated connective-tissue contents of the osteophyte spaces is apt to take place abruptly in the gap, which suggests that perforation has been effected from the osteophyte.

c. When the osteophyte ceases to advance, it becomes limited by a bony boundary, which is covered by cartilage, or, if the osteophyte reaches the surface, by a layer of fibrous tissue derived from the cartilage, and continuous eventually with the periosteum.

d. As the osteophyte grows, spaces form in its base by resorption, and as they enlarge the cancellous tissue and spaces of the osteophyte blend with those of the parent bone.
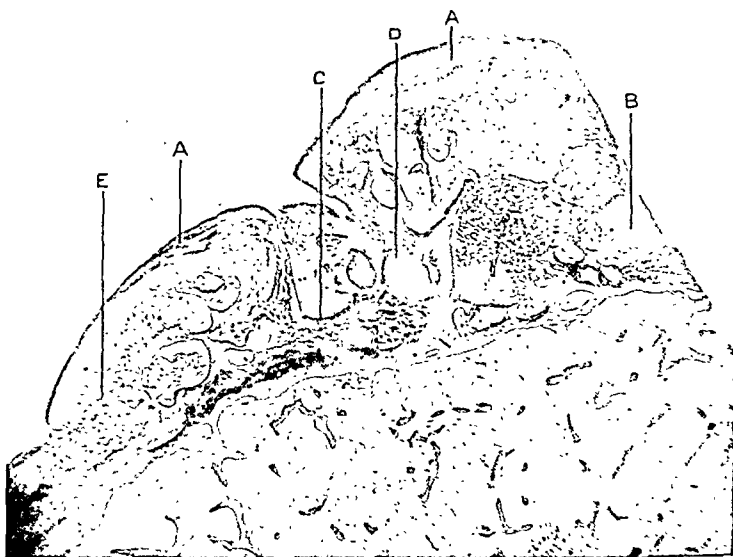
FIG. 149.—Osteo-arthritis. Section through a lumpy part of the articular surface of the head of a humerus of an old man. It was overlaid by the tendons of the spinati muscles and capsule, and it shows hypertrophic formations in various stages. A, Degenerating cartilage ; B, Granulation tissue ; C, Vascular fibrous tissue—cartilage transformed in the dividing sulcus; D, Granulation tissue eroding bone; E, Advancing ossification into cartilage. (*Specimen S.C. 15–1.*) (× 6.)

e. With the cessation of growth the fibrocellular contents tend to become fat marrow ; but in many microscope sections activity is seen to persist at some part of the osteophytic periphery. (*Inflammatory and Toxic Diseases of Bone,* Figs. 72, 74–77, pp. 151 et seq.)

Spaces, apparently identical with those just described as being the forerunners of an osteophyte in osteo-arthritis, occur in rheumatoid arthritis, but in the latter affection they show a more pronounced partiality for the neighbourhood where the cartilage joins the bone.

Up to this point the change is common to both disorders, and I assume the same cause to be at work—namely, toxins. But beyond this point the pathological processes in the two affections progress upon divergent lines. Localized ossification, tending to the building up of osteophytes or their homologues, produces the hypertrophic formations of osteo-arthritis ; but in rheumatoid arthritis inflammatory degeneration ends in the replacement of both bone and cartilage by fibrous tissue. These different responses to the

toxic damage are almost certainly determined by differences in the vitality of the tissues.

2. **Certain Peculiarities of the Calcified Zone of the Osteo-arthritic Joint.—** Histological features of some interest are associated with the development of

FIG. 150. — Osteo-arthritis. Section from the same femur and condyle as *Fig.* 156, showing an epiarticular osteophyte (B) near the centre of the transverse section of the condyle. A, Point of commencing bifurcation of the calcified zone. Note the bone on either side of the continuation of the normal calcified line. (× 8.)

osteophytes. Their occurrence, however, is only occasional. In the Strange-ways' Manuscript a bifurcation of the calcified layer of the articular cartilage
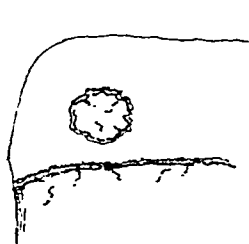
FIG. 151.
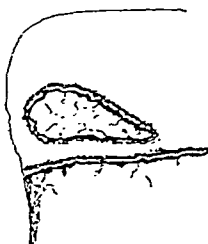
FIG. 152.
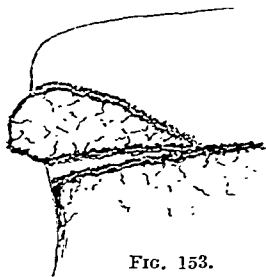
FIG. 153.
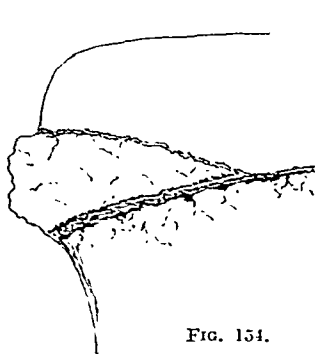
FIG. 154.
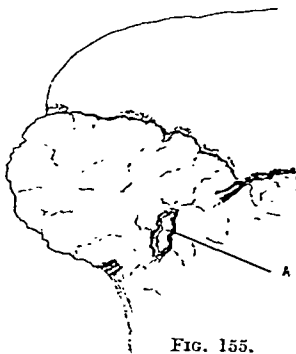
FIG. 155.

FIGS. 151–155. Diagrams to explain: (1) The bifurcation of the calcifying zone; and (2) The sequestration of pieces of cartilage (see the text). The inner black line = bone. The red line = calcified cartilage. *Fig.* 155 shows how traces of the basal line can be recognized in narrow trabeculæ showing a core of calcified cartilage; and also how a buried portion of articular cartilage (A) may arise (compare *Fig.* 153 and *Fig.* 157) and be left as an inset in an area of cancellous tissue.

at its marginal end is noted and illustrated more than once. At first sight it was not easy to understand how this duplication arose, but the examination

of a number of sections in which it appeared supplied the explanation. The manner of its development can be demonstrated best by the help of diagrams.

a. We may assume that an actively growing osteophyte forms in an articular cartilage not far from its margin. The osteophyte is surrounded by a zone of calcified cartilage, inside which is an osseous layer. The two represent the ossification zone of the growing nodule, whose cancellous structure connects with the bony layer containing it. A little distance below the osteophyte is the normal calcified layer of the articular cartilage lying upon the subchondral layer of bone. (Fig. 151.)

b. The osteophyte tends to grow in the direction of least resistance, and the pressure upon the articular surface helps to mould its shape, and direct it towards the margin. It assumes, often, a wedge shape, and the base of the wedge eventually projects at the side of the cartilage. Within the cartilage the osteophyte is still surrounded by its zone of chondral ossification. At this stage two calcified layers, separated by some of the deep layers of the articular cartilage, face one another—the upper one derived from the inferior surface of the osteophyte, the lower one being the marginal portion of the normal calcified zone. (Figs. 152, 153.)

Fig. 156.—Osteo-arthritis. An intracartilaginous osteophyte becoming marginal. From an external condyle of a femur. The bifurcation of the calcified zone is shown. A, Line of calcification above the osteophyte; B, The continuation of the normal calcified line. (Specimen S.C. 8.) (× 4.)

c. Advancing calcification absorbs the intervening cartilage, and the two calcified layers merge.

d. When this takes place the appearance of marginal bifurcation of the normal calcified layer is produced; the lower limb of the bifurcation is derived from the inferior surface of the osteophyte, which has blended with the end portion of the normal zone of calcification, and the upper limb is the ossifying zone of the upper surface of the osteophyte. (Fig. 154.) The lower limb is consequently composed of a calcified core sandwiched between two layers of bone.* (Fig. 150.)

The Collection contains a section from a proximal phalangeal joint (Micro. Slide S.C. 66–1) which shows one of those rugged hypertrophies that so often deform the articular ends of the metacarpal bones and phalanges in cases of osteo-arthritis. In the mass of new bone a calcified layer meanders in a very confusing way. The clue to its wanderings is to be found in this tendency of the calcified layer to divide and enclose an osteophyte. By tracing the calcified line in question in its bifurcations and ramifications, it is appreciated that these masses are formed by an agglomeration of osteophytes, which spring from the articular edge or the bone in its vicinity, and of secondary ones which develop from, and fuse with, those first formed.

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* See Inflammatory and Toxic Diseases of Bone, p. 140, and Specimen 810, St. Thomas's Hospital Museum.

3. **Buried Islands of Articular Cartilage.**—Another occasional outcome of the process just described is the sequestration of portions of articular cartilage in the cancellous tissue. An island of cartilage surrounded by an osseous border growing at its expense may be found amongst the cancellous tissue of an osteophyte, or of an articular bone-end, especially when osteophytic production runs riot upon its surface. Such an isolated piece of cartilage arises in this way. A portion of the chondro-osseous wall separating
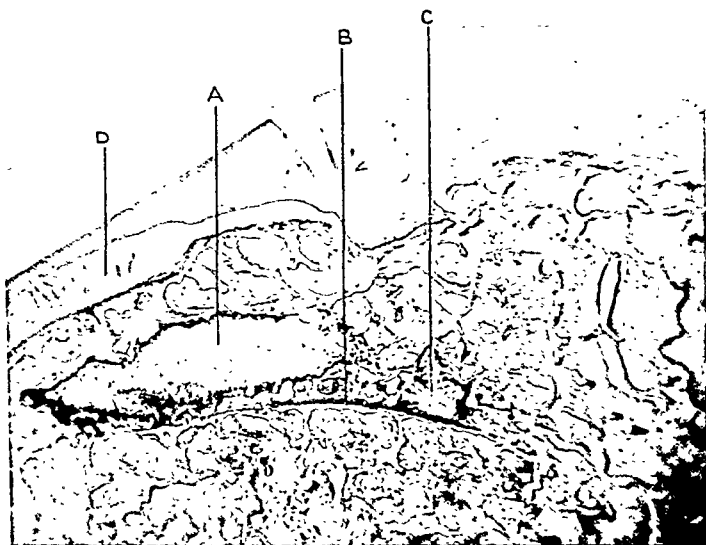
Fig. 157.—Osteo-arthritis—buried cartilage. From the posterior part of the internal condyle of the same knee from which *Figs.* 156 and 150 were taken. The surface at this point was markedly nodular. A, A buried portion of articular cartilage, ossifying on both sides, marks an inferior portion of the original cartilage ; B, The original calcified layer above which a buried epiarticular osteophyte is developing and cutting off C, an isolated portion, from A ; D, The superficial portion of the original articular cartilage with a calcified layer, covering the intracartilaginous osteophyte within the bounds of which the buried cartilage lies.

an osteophyte from the articular end of a bone is set free owing to resorption at its extremities. It is left *in situ*, but by the upgrowth of the cancellous tissue of the osteophyte it becomes surrounded by medullary spaces. It may be incorporated with the osseous framework in its vicinity and may lie at some depth from the articular surface. As the process of ossification continues such a submerged island must eventually disappear (*see Fig.* 155). (*Specimens* S.C. 1 ; S.C. 8.)

Another variety arises from the crumpling up—no doubt as the result of compression—of the articular cartilage overlying an area of extreme rarefaction. A detached portion becomes submerged. Such buried portions can be distinguished from the first form by their rugged and crushed-up appearance and the atrophic and inflammatory conditions in their vicinity. They, likewise, have a surrounding layer of bone. There are two examples in the microscopical preparations of the Collection.

B. Degenerative Changes in Sclerosed and Eburnated Bone.

Degenerative changes in eburnated bone may be studied in osteo-arthritic joints, and in those cases of rheumatoid arthritis which I have described as 'mixed'. The description of these changes may be preceded by that of some points in the histology of sclerosis, which, in the light they throw upon the changes seen in degeneration, are suggestive.

In the osteo-arthritic joint, when the articular surface retains a cartilaginous covering, sclerosis is apt to be slight, or absent, below it; and proliferation of the connective tissue in the underlying medullary spaces is either ill-marked or does not occur. On the other hand, where the cartilage

FIG. 158.—Osteo-arthritis. Section through an eburnated area of the head of the same humerus shown in *Fig*. 149. A, Thick eburnated calcified layer ; B, The thin subchondroid bone layer. There is absolute quiescence at the ossification area, and the marrow is composed of fat up to the bone. (*Specimen S.C.* 15–1.) (× 16.)
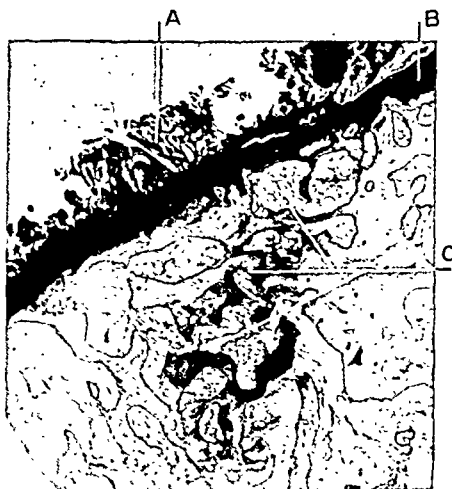
FIG. 159.—Osteo-arthritis showing degenerative changes. Section from a patella from the same case as *Figs*. 149 and 158. A, Arborescent cartilage; B, Eburnated calcified layer ; C, Shaded area marking the part where active degeneration and fat formation and disintegration of sclerosed bone are in progress. There are large numbers of good-sized vessels at this part. The upper limb of C points to a part which is shown more highly magnified in *Fig*. 160. (*Specimen S.C.* 15.) (× 14.)

has been worn away, and the calcified layer or the bone exposed, the surface is dense, often sclerosed to some depth, and the proliferation of the connective tissue in the spaces contained in it and in the adjacent medullary spaces is marked. Much of the new bone which is formed in the sclerosing process is produced by metaplasia of the vascular fibrocellular connective tissue with which the spaces in the sclerosing area are filled. The method of ossification explains why some of the bony masses which form the irregular mosaic of eburnated bone sometimes contain no central canal.

Now, a solid mass of compact bone badly supplied with blood-vessels cannot be very adequately nourished ; consequently, with the failure of vitality in old age or from ill health or other cause, the sclerosed bone soon

begins to show signs of degeneration. Here, a brief outline only can be given of what actually takes place.

The change begins with degeneration of small areas in the sclerosed substance. First, a group of cells becomes conspicuous in places where the bone looks granular and has lost its lamination. In a later stage a space has formed, which is filled with fibrocellular tissue; and this tissue blends with
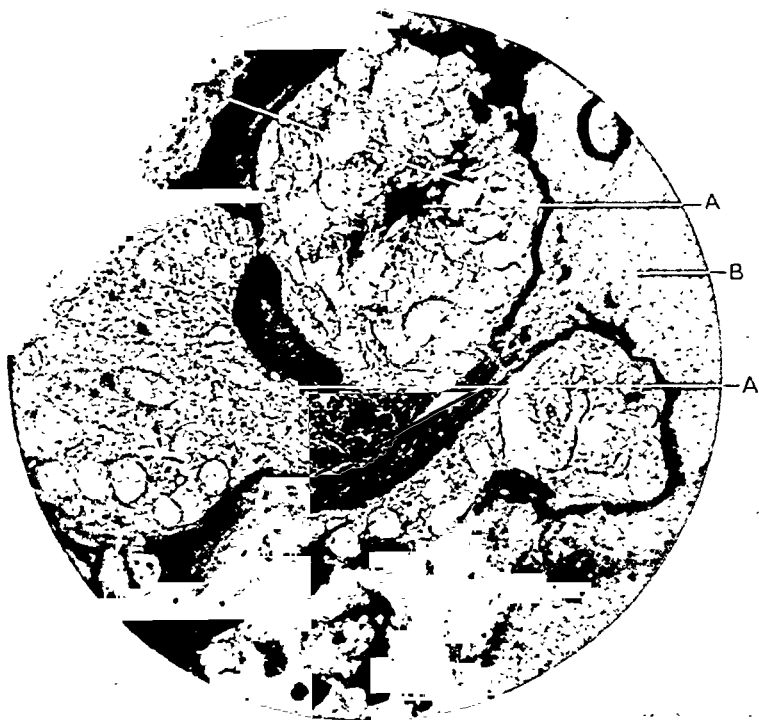
FIG. 160.—Osteo-arthritis—degeneration of bone. A more highly magnified portion of Fig. 159. A, Bone resolving into fibrous tissue. Contents of space, fibrous tissue, connective tissue and cells, and fat cells. B, Granular sclerotic bone, lamination lost or obscured in different parts. (× 75.)

a fibrillation which is taking place in some parts of the adjacent bone. As the sclerosed bone becomes honeycombed with such spaces, it gradually gets broken up into isolated portions, in which further progress of the resolution of the bone into fibrous tissue can usually be traced. The cellular elements of the disintegrating bone become connective tissue, forming a fibrocellular material in which the atrophying trabeculæ lie. Finally this tissue becomes fat medulla.

The eburnated articular surface of macerated bones are often seen to be marked with small 'pits' as if they had been pierced by a fairly large pin. These pits might possibly, but only for a moment, be mistaken for vascular apertures, : microscopically they are seen to be small areas, or surface clefts,

filled with fibrocellular tissue which has resulted from degeneration of the bone. The presence of these 'pits' indicates that an articular surface is entering on the degenerative stage, and that changes are beginning which, in the 'mixed cases', advance to such an extent as to graft a rheumatoid arthritic character upon an erstwhile osteo-arthritic joint.

Rarefactive atrophy, indeed, is frequently present in the hypertrophic formations of osteo-arthritic joints of elderly folks. A large marginal lip may be covered by a more or less eroded cortex enclosing a very wide-meshed and delicate cancellous interior. This atrophic condition is due in most cases to the fibrous form of bone degeneration, though there are exceptions. All the osteo-arthritic specimens in the collection came from patients whose ages varied from 63 to 83. The fibrous form of degenerative atrophy was found in all but one of the specimens in which the condition could be microscopically checked. Moreover, degeneration varied in degree in different cases, and in the oldest—a woman aged 83—it was only beginning.

The parts specially liable to degeneration were the osteophytes, and the eburnated and sclerosed areas—that is, the more recently formed bone— seemed the most prone to suffer. It will be noted that the bone selected was that which, in the main, resulted from the metaplasic process of ossification.

II. RHEUMATOID ARTHRITIS.

The category of rheumatoid arthritis comprises cases which end in bony ankylosis, cases in which fibrous ankylosis is the rule, and many others in which ankylosis never occurs, probably because the disease has not had time to run its course, or because the intensity of the disorder has been too slight.

The difference in the nature of these two forms of ankylosis has been explained in Part I; how, in the first case, articular cartilages are stimulated to changes that tend to ossification, and, in the second, cartilage and bone undergo degeneration. The cases in the Strangeways Collection in which bony ankylosis of several large joints occurs are few; but those in which fibrous union develops form a large majority; also the onset of degeneration may transform the type that ankyloses by bone into that which ends in fibrous union.

An illustration of this is provided by a knee-joint (S.C. 50–2) in which the femur and patella are united by an intervening band of tissue partly cartilaginous, but largely fibrous. In the fibrous part an osseous bond had been established between the bones, but owing to degenerative changes involving the new bone as well as the bones which it joined, bony union has been interrupted, and the new trabecula changed into fibrocellular tissue. In this case the patient was a woman in whom the disease began at 19, and death occurred at the age of 37. (*See also* S.C. 60–1.) These facts tend to show that *the pathological process which is specially to be identified with rheumatoid arthritis is degeneration — a degeneration in which both articular cartilage and bone are replaced by fibrous tissue.*

The line between progressive and degenerative change in rheumatoid arthritis is not sharply drawn and mixed conditions may be present. Thus in the very case from which the changes leading up to bony ankylosis were

described, there are also some signs of degeneration in both the cartilage and the bone (S.C. 27). It will be remembered that in that case only two joints out of the eight included in the Collection were not the subject of bony ankylosis. When the patient died at the age of 54 those two joints were hovering between tissue growth and tissue degeneration. Had she lived much longer, it is not unlikely that the latter process would have gained the upper hand, and the degenerative form of rheumatoid arthritis and fibrous ankylosis have resulted.

Sometimes one joint does become ankylosed by bone and another by fibrous tissue; for instance, in a woman of 71, the right knee was fixed by bony union, and the left was the subject of fibrous ankylosis. Fibrous degeneration of the articular bone was so pronounced in the latter joint that
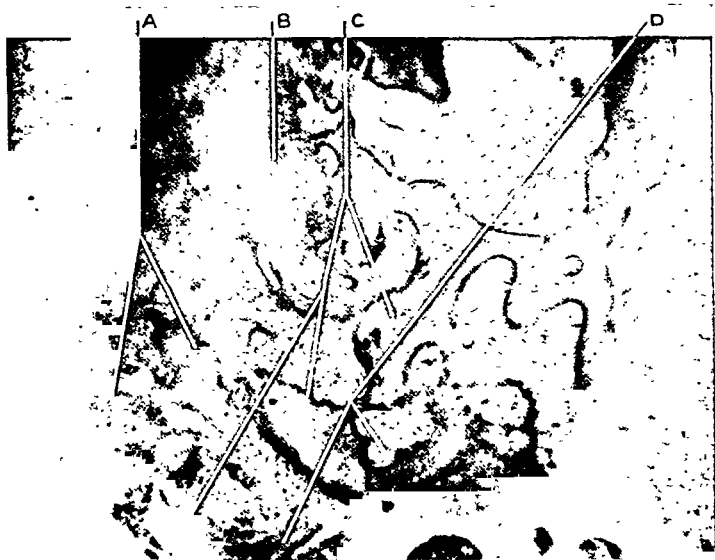
FIG. 161.—Rheumatoid arthritis. Section from the posterior part of the same condyle as that shown in *Fig.* 162. It shows the pronounced changes so commonly seen in rheumatoid arthritis at the junction of the cartilage and bone, and how both calcified cartilage and bone tend to degenerate into fibrous tissue. A, Zone of calcified cartilage; B, Fibrocartilage lying on bone, apparently replacing the calcified cartilage; C, Bone trabeculæ undergoing fibrous resolution at their margins. D, Spaces filled with fibrocellular tissue—no inflammatory cells. (× 50.)

no bony union would have been possible. (S.C. 26; S.C. 26–1.) Such a case may point to unequal powers of resistance in the two joints or to a failure of general joint vitality after bony ankylosis has already occurred in one.

It has been shown that fibrous degeneration of cartilage and bone is not a purely senile change (Part I, pp. 128, 129). Nor is it peculiar to the rheumatoid affections, for it was present in a chronic septic knee amputated at a war hospital in England. But it is found in all forms of chronic rheumatoid disease.

In osteo-arthritis it is present in many cases well advanced in years (*see* p. 316). In rheumatoid arthritis the bony ankylosing type may, in its later stages, show signs of it; whilst in the cases that end in fibrous ankylosis, fibrous degeneration of bone and cartilage is the outstanding histological feature and may or may not be associated with marked signs of inflammation.
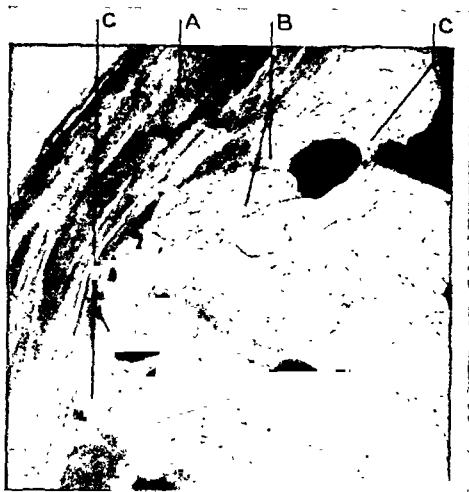
FIG. 162.—Rheumatoid arthritis. Section from the anterior part of the internal condyle of the left knee of a woman aged 76. It shows the fibroid degeneration of the articular surface structures. A. Fibrous covering—partly transformed cartilage, partly resolved bone; B, Fibrous strands representing resolved bone; C, Resolving parts of edges of trabeculæ. (*Specimen S.C.* 41–5.) (× 50.)

In a further contribution (Part III) I shall show that the same form of degeneration is an important factor in the joint changes in Charcot's disease. (*See also Fig.* 41, p. 117, Part I.)

THE HISTOLOGY OF FIBROUS DEGENERATION OF
CARTILAGE AND BONE.

The form of degeneration taken by cartilage and bone in rheumatoid arthritis calls for a more detailed description than the fragmentary outline already given (p. 315). The histology is not simple and appearances are apt to be confusing. Some are very like changes seen in ossification, and it may not always be easy to decide whether they point to a stage in an atypical production of bone or to its degeneration. Indeed, often only by an appreciation of surrounding conditions can an observer be sure that he is looking at resolving and not growing bone, or that a space is a consequence of degeneration, and not a primitive medullary space engaged in preparing the way for ossification.

1. **The Formation of Spaces in Bone.**—The changes occurring in fibrous degeneration of bone may be studied in their initial stages in the central parts of a large trabecula or in a mass of sclerosed bone.

The first indication is a localized crowding of cells, and a granular

obscuration of the bone structure. The crowded cells are certainly not all bone-cells. Delicate fibres may sometimes be seen amongst them, and adjacent bone-cell cavities may appear too large for the cells they contain. The next stage is the appearance of a space filled with the cells supported in a mesh-work of fine fibrous tissue. The cells are now connective-tissue cells, fusiform, stellate, or angular; and, rarely, a bone-cell, which possibly has escaped modification, may be recognized. The bony margins of the space may show at some part a blurring, in which it is possible to focus numerous fine fibrils issuing from the bone. These fibrils are lost in the fibrocellular contents of the space. In a sclerosed mass there may be several
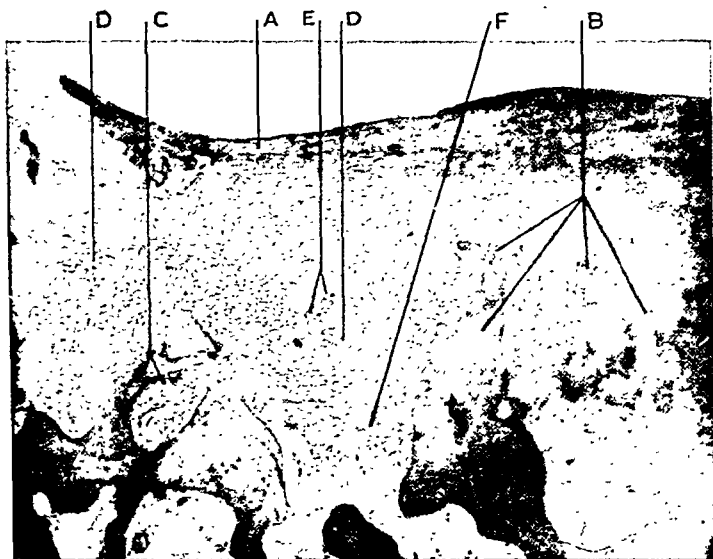
Fig. 163.—Rheumatoid arthritis. Section from the articular surface of the tibia from the same knee as *Figs.* 161 and 162. It shows destruction of the calcified cartilage and subchondral bone. A. Degenerating articular cartilage, superficial part ; B, Zone of calcified cartilage ; C. Trabeculæ undergoing resolution into fibrous tissue at their edges ; D. Thick layer of proliferated fibro-connective tissue containing disappearing portions of calcified cartilage ; E. Giant cells ; F. A trabecula almost completely resolved into fibrous tissue. (× 50.)

cellular foci or spaces of this nature, and other larger spaces with similar contents have clearly resulted from the confluence of several small ones. The subchondral layer of bone is a favourite situation for these spaces, and in some joints they may form quite a long row in it without trenching upon the calcified layer. (S.C. 49 from Kathleen M., age 16, is an instance.) Frequently, however, that layer, which is particularly prone to suffer in this form of degeneration, is also involved. Giant cells are very often a conspicuous feature in these spaces, and appear at an early stage in their development. They no doubt play an important part in their formation and enlargement (*see* p. 323).

2. **Fibrous Resolution of Bone.**—In addition to the formation of spaces,

the margins of trabeculæ are affected, and especially portions of the sub-chondral bone layer. The degenerative process is indicated by the thinning and often granular appearance of the bone, by an increase in prominence and number of the bone-cell cavities and their contained cells, and by an emergence of crowded fibrils to join the fibro-connective-tissue contents of the medullary spaces, in whose bordering trabeculæ this change is going on. Often bands of fibrous tissue, representing portions of vanished bone, may be seen bridging an interruption of the subchondral layer, linking up neigh-bouring trabeculæ or forming a portion of the peripheral laminæ of others. The bone in the vicinity of these fibrous joinings usually shows signs of degenerative change. (*Fig.* 162.)

Again, a thin layer—more cellular than fibrous—is not infrequently interposed between the cartilage and the subchondral layer of bone, and

FIG. 164.—Rheumatoid arthritis. Section from the articular surface of the head of the radius (right elbow) from the same patient as *Figs.* 161 to 163. It shows the fibrous degenera-tion of bone and cartilage at the articular surface. A. Fibrous covering made up of trans-formed cartilage and resolved bone; B. Fibrous villus on the surface; C, Trabeculæ undergoing fibrous resolution; D, Degeneration in the body of a trabecula; E, Vessels—inflammatory cells. (*Specimen S.C.* 41–7.) (× 50.)

contrasts strongly with both. The margin of the bone on which the layer rests is eroded, as if it had been nibbled by a mouse, an appearance which is produced by the opening up of the bone-cell spaces. In some places traces of degeneration are present in the interior of the bone. I have no doubt that this fibrocellular layer originates from the decaying bony artic-ular surface.

Resolution of bone into fibrous tissue may not only extend along the subchondral layer, but may involve the deeper trabeculæ in places, causing more or less destruction. Indeed, in some cases large tracts or masses of

fibrous tissue occupy the place of a number of trabeculæ, to whose previous existence small portions of resolving bone bear witness.

3. **Fibrous Resolution of Cartilage.**—In the cartilage very similar changes give rise to spaces filled with fibro-connective-tissue of the type already described. Frequently portions of cartilage, either lying isolated within these spaces or forming a portion of their walls, may be seen giving off a wisp of fibres which blend eventually with the contents of the space.

There is no danger of confusing the above changes with *the fibrous transformation of the articular cartilage* which is so often seen in rheumatoid arthritis.
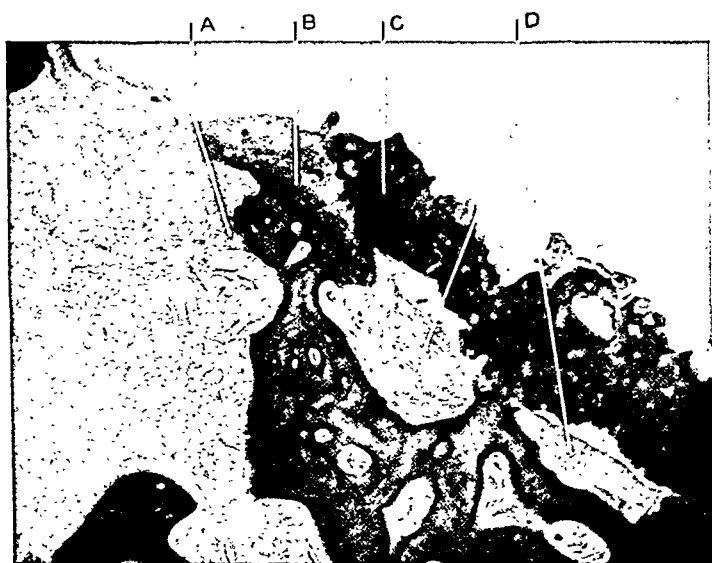
FIG. 165.—Rheumatoid arthritis. Section showing the articular surface of the ulna, from the same elbow as *Fig.* 164. A, Part of a trabecula resolving into fibrous tissue at its margin ; B, A portion of degenerate subchondroid bone layer giving off fibrous tissue at both ends · C, Degenerating cartilage—spaces contain proliferated connective tissue ; D, Spaces resulting from degeneration of large portions of the subchondral bone layer and adjacent cartilage, containing fibrous tissue, connective-tissue cells, vessels, and fading relics of bone tissue. At a few points on their margins the bone is giving off fine fibrous tissue. (*Specimen S.C.* 41–7.) (× 50.)

The latter usually starts at the surface as fine horizontal fibres in the substance of the cartilage, and goes on to fibrous replacement of the superficial layers. It may even involve the whole depth of the cartilage without disguising its identity. It is not necessary for it to be accompanied by the degenerative signs I am describing, but the two forms of fibrous change are often seen together.

Any difficulty in deciding as to the true character of the changes which have been specified will usually disappear upon a comprehensive review of the section. As a rule it is evident that bone and cartilage are steadily disappearing ; their distinctive characters are being lost, and they are in process of being replaced by fibrous tissue, which is sometimes very cellular
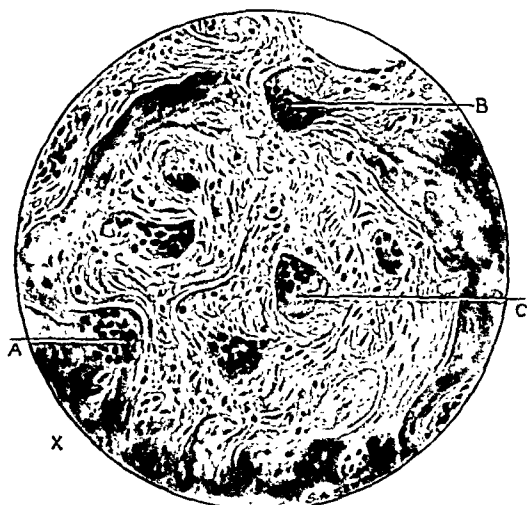
FIG. 166.—Origin of a giant cell in bone substance. Drawing from a section from the articular surface of the femur in a septic knee-joint, obtained from a war hospital in England, showing the formation of a giant cell in bone or possibly calcified cartilage. A, Giant cell forming in a projection of the subchondroid bone layer or the adjacent calcified zone; B, Another giant cell whose position and the appearance of surrounding parts suggest recent separation from the bone; C, Another with resolving bone attached; X indicates the position of the articular cartilage outside the picture. (*Specimen* S.C. 99.) (× 235.)
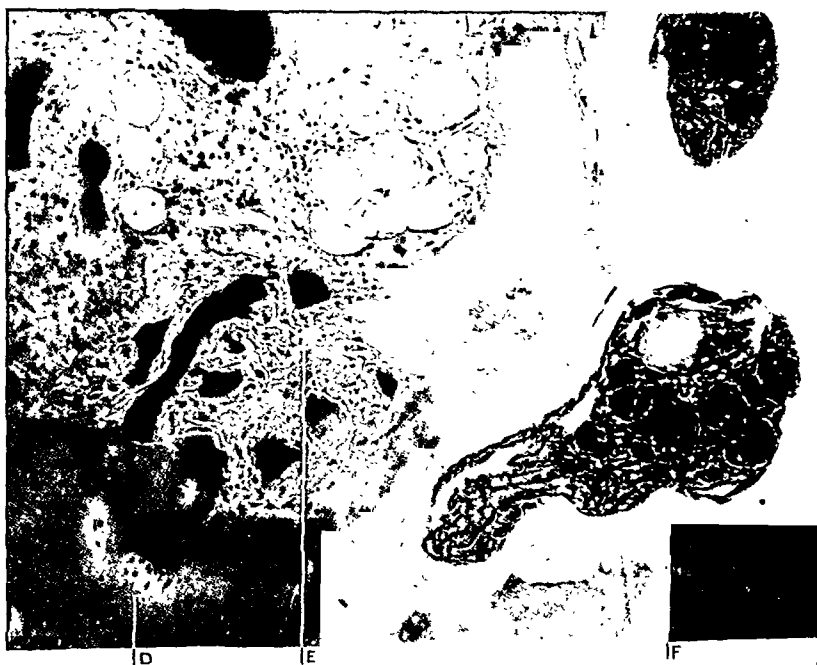
FIG. 167.—Photomicrograph from a section from the articular surface of the femur in a septic knee-joint. obtained from a war hospital in England, showing the formation of a giant cell in calcified cartilage or possibly bone. D. Articular cartilage; E, Portion shown in *Fig.* 166; F, Subchondral bone layer. (*Specimen* S.C. 99.) (× 120.)

and sometimes only slightly so. The changes are most marked in the imme-
diate neighbourhood of the cartilaginous junction, and in many cases quite
long tracts of the calcified layer, with adjacent portions of the cartilage and
the subchondral bone layer, have vanished.

The Origin of Giant Cells associated with Bone. —An important histo-
logical feature of these rheumatoid cases is the frequency with which giant
cells, often in considerable numbers, are found in the degenerating areas.
They excited no particular interest until one was found among an aggregation
of cells which heralded the formation of a space. It was then realized that
they were often present in the smaller recently formed spaces. Four or five
were not uncommon, and in a space formed by the coalescence of several
small ones more than twenty were counted. Further, it was found that a
giant cell was present in a potential space more commonly than had been
suspected, and the idea that they might prove to be formed from cells
within the substance of bone or cartilage occurred to me. The suspicion
was confirmed by a section cut from the femoral condyle of the chronic
septic joint (S.C. 99) already mentioned. In this slide two spaces bearing
the usual signs of fibrous degeneration of bone are present in the subchondral
layer beneath a strip of cartilage, which at this part has not been seriously
affected by the inflammatory process. On the margin of one of the spaces
is a nipple-like projection, and in its interior are some twenty or thirty
nuclei crowded together. At first taken for a giant cell, it was soon seen
to be part of the bone or calcified layer, which at this point are not readily
distinguishable from one another. On the deep attached side of this projec-
tion disintegrating changes are just beginning to take place and the multi-
nucleated mass would no doubt eventually become detached. There are six
other giant cells in the space ; one has evidently just separated from another
part of the margin, and another blends with a granular mass as big as
itself, whose fibrous interior and emanations point to its being a resolving
fragment of bone.*

Evidently in the fibrous degeneration which occurs in rheumatoid arthritis
the disintegration of bone and cartilage is materially helped by the develop-
ment of giant cells in their substance, and the solution of the matrix surrounding
them ; whilst the formation of such cells in the bone or cartilage substance,
and their subsequent separation, will cause a form of crumbling away of
tissue not hitherto envisaged.

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* The following observations by Professor M. J. Stewart are of interest in connection
with this matter :—

" The bone consists in the first place of osseous tissue proper, which includes bone
cells, calcified matrix, osteoblasts, and osteoclasts. These are enclosed in a sheath of
fibrous tissue, the most obvious and complete portion of which is periosteum. The inner
aspect of the bone, however, is also covered by a thinner and more delicate layer of fibrous
tissue—the endosteum—which not merely lines the main medullary cavity itself, but clothes
all the little bony lamellæ of the cancellous tissue . . . my own histological observations
have assured me that the osteoclast is met with only in relation to bone, and that it does
not occur throughout the marrow generally. Its normal position is within the bony tissue
or at least between the bone and its peri- or endosteum."

" Histogenesis of Myeloid Sarcoma ", *Lancet*, 1922, ii, 1906.

SPECIFIC FORMS OF RHEUMATOID ARTHRITIS.

The Collection contains a group of specimens obtained from cases which were infected with some specific disease. These cases were marked by an illness of many years' duration, by painful chronic swellings of numerous joints, and by a crippled and bed-ridden condition, which terminated in death from exhaustion. In their clinical course they resembled, and were looked upon as, rheumatoid arthritis; but one was subsequently proved to be due to pneumococcal infection; another is suspected to have been syphilitic (congenital) because of a general endo-arteritis affecting the synovial vessels; and of two others, one was due to the gonococcus, and in the second the gono-coccus was probably closely associated with the latter part of its course. Fibrous degeneration of bone, like that seen in rheumatoid arthritis, was found in all of them.

The clinical course and histology justify these cases being regarded as rheumatoid arthritis due to a specific cause—an infection. The type is quite distinct from the well-recognized joint conditions commonly attributed to such infections; and the specific rheumatoid form may easily be distinguished by the use of the suitable adjective, e.g., pneumococcal, syphilitic, gonorrhœal, rheumatoid arthritis. There would be much advantage in such a nomen-clature, for since these cases are usually considered to be rheumatoid arthritis during life, a correct diagnosis is more likely to be made, if it is taught and appreciated that such specific types occur.

Case 7.—Pneumococcal rheumatoid arthritis. (*Specimens S.C. 57 Group.*)

John S., a carman, age 31. Some years before death joint trouble began grad-ually with swelling and a little pain in the right knee. These increased and he was laid up for thirteen weeks. Then the swel-ling subsided, and he worked for two years. In 1906 the right knee again became swol-len, and he was laid up for three months. In 1907 both knees became swollen and painful, and he never worked again. In 1908 he contracted gonorrhœa, which cleared up without apparently affecting any joint. In December he had iritis, and though he was bedridden the right ankle became swollen and painful. In November, 1909, he entered St. Pancras Infirmary. Both knees were swollen and contained fluid. Temperature and urine were normal. During 1910 he was more or less in bed with recurring trouble in knees, ankles, and joints of the upper ex-tremities. In 1911 Dr. Strangeways saw him and noted that he was a "case of apparently typical rheumatoid arthritis with great deformity and pain in the joints and limbs. There were spindle-shaped swellings of the phalangeal joints." Fur-ther attacks of arthritis occurred in September, followed by iritis, and then more arthritis. In January to April, 1912, there was more iritis, and the left eye was excised. In July he became worse with right-sided pleurisy, and died.
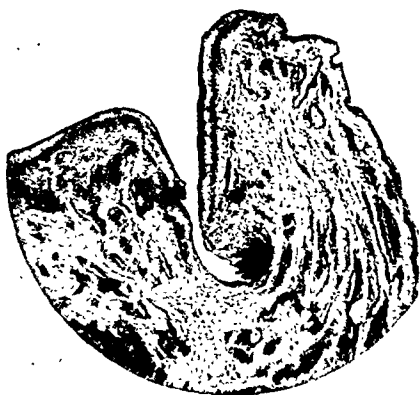
FIG. 168.—Pneumococcal rheumatoid ar-thritis. Section through a portion of the capsule of the right shoulder. Showing a villous process and leucocytic infiltration: from a man aged 31. (*Specimen S.C. 57.*) (× 25.)

´Post´ Mortem.—Turbid greenish pus with a foul odour was found in both shoulders and both elbows. A pure culture of a Gram-positive diplococcus was obtained from the pus, and reproduced after animal inoculation. This was authoritatively stated to be undoubtedly pneumococcal. A Gram-positive diplococcus was also demonstrated in sections from the capsule of the knee—at the College of Surgeons.

Microscopical Examination.—Examination of slides from the shoulder and elbow showed marked evidence of inflammation and destruction of tissue in the peripheral part of the articular surface. The destruction involved the deep portion of the articular cartilage, the calcified zone, and the subchondral layer, and was due
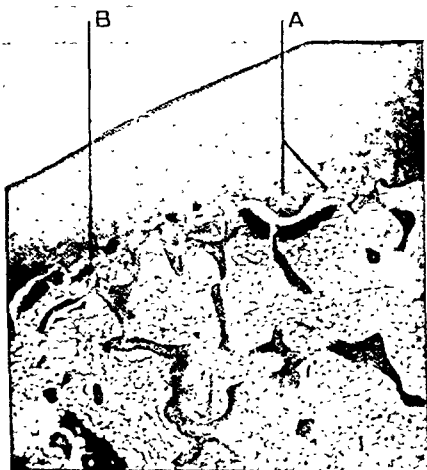
FIG. 169.—Pneumococcal rheumatoid arthritis. Section from the head of the right humerus from the same patient as *Fig.* 168, showing degeneration and destruction of the subchondroid bone layer and the zone of calcification beneath practically normal cartilage. A. Degenerating spaces in the subchondral bone layer ; B. At peripheral part of the articular surface the calcified layer, parts of the deep layer of the cartilage, and portions of the subchondral bone layer have disappeared. (× 14.)
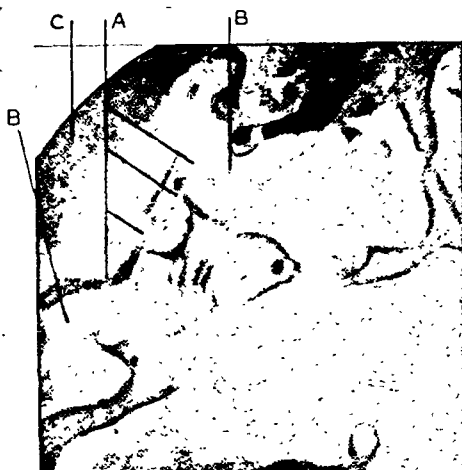
FIG. 170.—Pneumococcal rheumatoid arthritis. From the same section as *Fig.* 169 (humerus), but nearer the articular margin. It shows the inflammatory tissue in the medulla, granulation tissue, vessels, fibrous tissue, diffuse inflammatory cell infiltration, also the disappearance of the subchondral bone layer. A. Bone degenerating and resolving into fibrous tissue ; B. Granulation tissue ; C. Articular cartilage. (× 40.)

to the degeneration of bone and cartilage into fibrocellular tissue. Characteristic degenerative spaces were present in central parts of the trabeculæ of the subchondral bone layer and there was abundant evidence of fibrous resolution of bone. The adjacent periosteum was inflamed.

(T. S. P. Strangeways, *Bulletin Camb. Com. for the Study of Special Diseases,* iv, No. 6, 87.)

The possibility that in this case the pneumococcal infection was superimposed on rheumatoid arthritis has to be taken into account.

Case 8.—Syphilitic (congenital) rheumatoid arthritis.

A young man, age 22, had his right knee-joint excised by Mr. Wherry in Addenbrooke's Hospital, Cambridge, for a condition thought to be tuberculous. There was a history of two years' pain. swelling, and resistance to treatment. He made an uneventful recovery from the operation, but after leaving the hospital all his joints became affected, and in due course he was completely crippled. There was pain, swelling, and stiffness especially in the hips, left knee, ankles, and hands, and he could not turn in bed. His shoulders were less involved. The hands were deformed.

There were no discharging sinuses connected with the joints. At the age of 33 he developed pneumonia, and not long after died from exhaustion caused by bed-sores.

A sister is stated to have suffered for about forty years from tuberculous disease of both feet. The right foot was amputated in 1913; and the left, which had discharging sinuses, in 1916. No attempt was made to substantiate the diagnosis at the time (personal communication).

The specimen (S.C. 58), which consists of the parts removed by excision, is remarkable for the great thickening of the synovial membrane, and its villous projections, which have a lumpy tuberous form. Microscopically there is no evidence of tubercle,
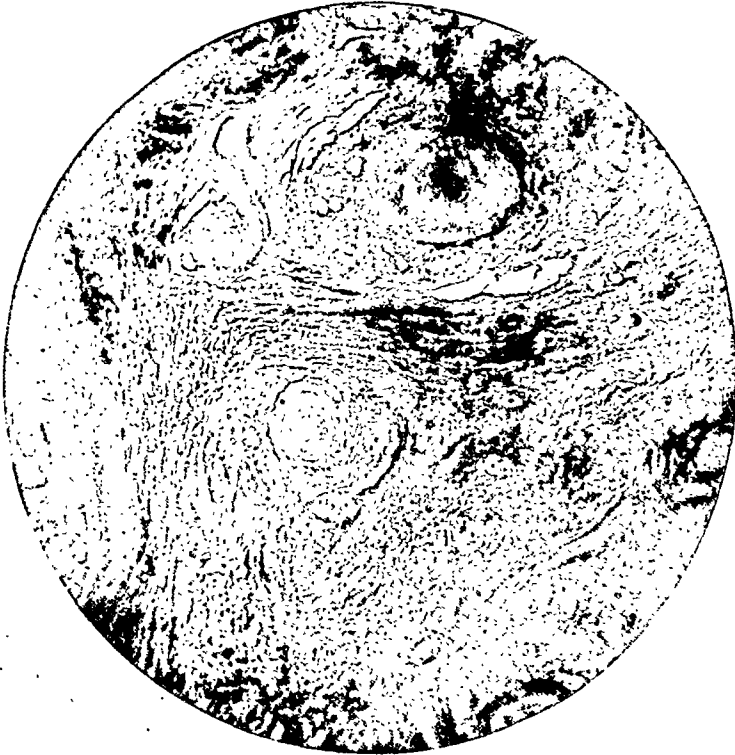
Fig. 171.—Syphilitic rheumatoid arthritis. Section of chronically inflamed synovial membrane from a knee-joint of *Case* 8, showing endo-arteritis of vessels. (*Specimen* S.C. 58.) (× 40.)

but the blood-vessels of the synovial membrane are remarkable. They are very numerous, and the subject of chronic endo-arteritis, which has obliterated the lumen of many. The peculiar condition of the blood-vessels is believed to point definitely to the syphilitic nature of the disease. There are pronounced changes in the trabeculæ, and in parts of the articular cartilage of the femur. The subarticular bone is generally involved, and where the cartilage has disappeared the destruction is marked. Resolution into fibrous tissue, and degenerative spaces, occur. In many places the former is very beautifully shown, both in the cortex of the side of the condyle beneath the thickened capsule, and in the part referred to above where the cartilage is absent. This form of degeneration is less noticeable in the patella, where atrophy of the cancellous tissue is pronounced.

Search was made for spirochætes and tubercle bacilli in the synovial membrane taken from the specimen, but neither were found. (R.C.S.)

Case 9.—Gonorrhœal rheumatoid arthritis.

W. A., a wood turner, who was 48 years old when he died, contracted gonorrhœa at 19, had rheumatic fever at 21, and again when he was 29 in 1903. Subsequently he was subject to pains all over, but particularly down his spine. In 1906 the right shoulder and elbow became increasingly stiff, and later the left shoulder and elbow were attacked. In 1908 the spine was involved, and in 1909 the jaw. In this year he had to give up working. The right knee (S.C. 61) became swollen in 1910. The patient became very emaciated and his weight fell to 6 st. 9 lb. There was much muscular wasting, diagnosed as progressive muscular atrophy, but there were " none of the characteristic changes associated with osteo-arthritis." He died from bronchitis in 1911.

POST MORTEM.—The knees, hips, and elbows were flexed, and movement was only possible through an extremely limited range. The ankles, shoulders, and wrists were in a similar condition. The hands were contracted, and the small muscles, like the general musculature, were in a condition of advanced atrophy. Gonococci were found in a portion of the capsule taken from the specimen (R.C.S.). Microscopically, a section from the femoral condyle shows pronounced signs of degeneration in the form of fibrous resolution. This occurs in the neighbourhood of a sharp-edged marginal ulceration of the articular surface. The floor of the ulcer is formed by granulation tissue which penetrates amongst the cancelli below. A similar, but a small, area of degeneration is seen at some distance from the margin, where an erosion of the cartilage has exposed the subarticular bone. Besides this there are a few characteristic degenerative spaces with fibrous margins distributed in the cartilage and in the subchondral bone layer. (*Fig.* 172.)

A peculiar appearance of the fat marrow is present in this case. The fat cells have a protoplasmic mass of variable size situated at some part of their periphery. The fat cells themselves are not as a rule in contact, but are surrounded by a clear ring-like space, and occasionally the retiform connective tissue can be seen to have its spaces distended. These appearances point to fat absorption; the pressure of the œdematous fluid, whose presence can be explained

FIG. 172.—Gonorrhœal rheumatoid arthritis: part of the articular surface of the internal condyle from the knee-joint of *Case* 9. A. Spaces resulting from bone degeneration, with fibrocellular contents, giant cells, and degenerating margins: B. Granulation tissue; C. Surface fibrous covering, largely the result of cartilage degeneration; D. A collection of inflammatory cells. Fibrous resolution of bone is to be seen in places. (*Specimen* S.C. 61.) (× 25.)

by the inflamed state of the medulla, may be an auxiliary factor. (*Fig.* 173.)

A similar condition was met with in two other cases (S.C. 31, S.C. 55) where emaciation was advanced, but in many others apparently equally wasted the fat marrow was natural.

Case 10.—Gonorrhœal rheumatoid arthritis.

A man who was 38 when he died in 1912 had an illness, diagnosed as 'gastric fever', in 1887. At the age of 19 (1893) he was ill for fifteen weeks with severe rheumatic fever. At 21 (1895) he contracted gonorrhœa, which lasted three months and cleared up. In 1898 he was ill for eight months with rheumatism in the hips and knees. From this he recovered, but in 1903 he developed acute pain in the left knee and hips, and was two months in University College Hospital. In 1912—the year of his death—he had been bedridden for years. The hands were much distorted, the spine was ankylosed and curved, and the movements of all the joints were greatly limited.

Doubtful gonococci were found in the capsule of the knee-joint (S.C. 60). The cocci were Gram-negative; in places they were diplococci; some were intracellular, but their shape was not distinctive (R.C.S.). Microscopically, degeneration of cartilage and bone into fibrocellular tissue, like that seen in rheumatoid arthritis, is present. There is, also, much inflammation. In the fibrous union of the patella to the femur there are signs of an attempt at bony ankylosis, which had been frustrated by extensive degeneration of the bone and cartilage.
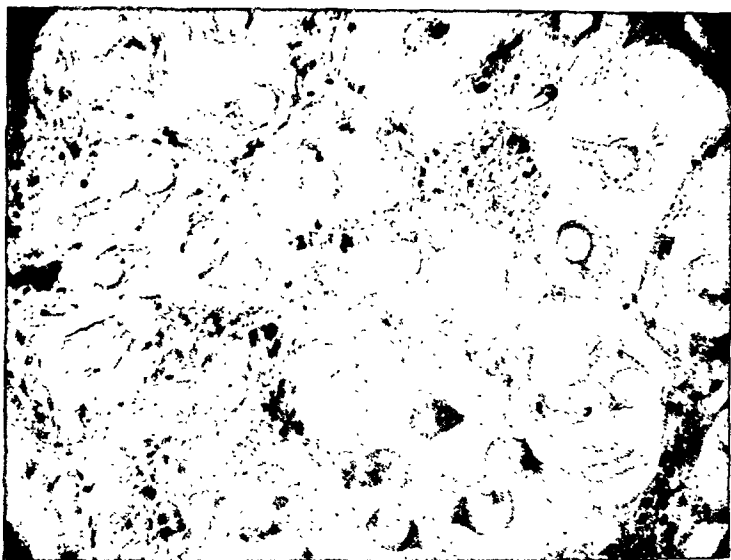
FIG. 173.—Gonorrhœal rheumatoid arthritis. From the same section of the internal condyle as *Fig.* 172. To show the œdematous fat medulla; the fat globules undergoing absorption. (× 200.)

In this instance the fibrous form of ankylosis developed in a man who had shown rheumatic tendencies before he contracted gonorrhœa. The picture may be completed by reference to a case that I have recorded in which generalized bony ankylosis followed gonorrhœa in a gentleman, age 37, who some time before he contracted gonorrhœa had suffered from pleuritic effusion, and had developed effusion into both knees whilst convalescing from this illness. (*The Inflammatory and Toxic Diseases of Bone*, p. 182, Case 5.)

SCHULLER'S VILLOUS ARTHRITIS.

Case 11.—This case is placed after those illustrating specific forms of rheumatoid arthritis because there is not sufficient justification for including it in that group. Though little more than a fragment, it is recorded here because, clinically, it seems to belong to the rheumatoid arthritis category, and as similar cases are uncommon it may be useful as well as interesting. There is only one specimen of this condition in the Collection (S.C. 53). Unfortunately it comprises only a few of the villous processes; and the termination of the case could not be traced. The exhibit consists of several more or less pedunculated, soft, lobulated masses resembling in appearance portions of parotid gland.

Microscopical Examination.—A slightly magnified section of one of the masses is reminiscent of a rectal polypus in the arrangement of its lobules. Under the usual powers the interior presents an anastomosing framework of fibrous tissue forming numerous spaces, in which are sections of vessels and a reticular stroma supporting connective-tissue cells more or less crowded. These cells have large nuclei and a narrow cell-substance, and some larger ones have more than one nucleus.

In some lobules the cells are pressed together and have a polyangular shape, whilst the fibrous framework is more defined. The containing border of a lobule is formed by a layer of rather dense fibrous tissue, in which are oval cells of considerable size with large pale-staining nuclei. Situated in the cellular parts of the interior are a few isolated areas of crowded inflammatory cells ; they contain no giant cells. Amongst some of them are narrow tracts of cells with large nuclei, not so deeply stained as the inflammatory cells, or as the connective-tissue cells of the parenchyma. They are possibly endothelial cells—derived from vessels, lymphatic or otherwise. A striking feature is the great number of vessels in cross-section, each occupying a central portion in a space. These, and the scattered fat cells. and the loose arrangement of the parenchymatous cells, give the impression of a somewhat open texture. No tubercle bacilli were found in a recent examination, and no other organisms were noted.

CLINICAL HISTORY.—The patient was a motor-lorry driver. and was consequently much exposed to wet and cold in his occupation, but he was always fit and hard like other men. He worked from fifteen to twenty hours a day (*sic*). used to weigh
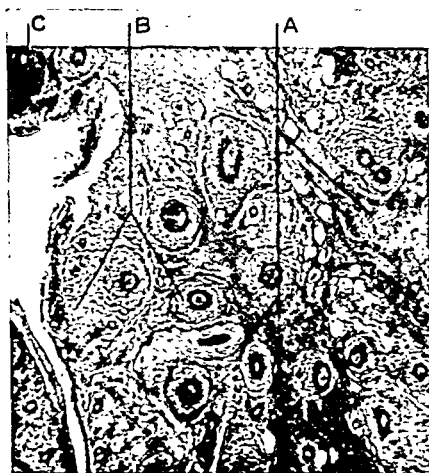
Fig. 174.—Schuller's villous arthritis. Section through a polypoid villus. A. Fibrous framework : B. Connective-tissue cells ; C, Inflammatory cell groups. (*Specimen* S.C. 53.) (× 50.)

13 stone. but had lost flesh since his illness began. He looked pale, but never felt ill or depressed. The first sign of trouble began in July. 1921. when a gradually increasing stiffness in the left arm came on when he was driving. There had been

no previous injury. Swelling of the knee followed in August and slowly increased. In two or three months it reached the size that persisted whilst he was under observation. Pain at first was slight and he was just able to get about. It got worse, but was never severe, and after a few months he was admitted to the London Hospital. There was a similar history for the right knee. The left ankle became affected in November, and the left elbow about the same time. One wrist followed in March, 1922, and the right shoulder towards the end of the year. In Marylebone Hospital " the right knee was aspirated on two occasions. No organism was ever obtained on culture of the fluid." A radiogram showed " no bony changes, approximation of joint lines, and thickening and swelling of the soft structures of the joint." " On June 9, 1922, a small portion of villous growth was removed at operation " (arthrotomy). " Mr. Strangeways diagnosed Schuller's villous arthritis. At that time the patient had tubercle bacilli present in the sputum on many occasions, and the (lung) disease was fairly extensive." He was re-admitted on Sept. 12, 1923, with his joints not materially altered, and was transferred to West Ham Hospital in February, 1924. It is impossible to ascertain his history beyond this point, but " in view of the lung condition in 1922 it is improbable that he is still alive."*

Max Schuller, who first described the condition, and found cocci and bacilli in the villous processes, calls it " inflammatory hyperplasia of the normal synovial processes as they occur in chronically inflamed synovial membranes " (*Arch. f. klin. Chir.*, 1893, xlv, 153). His illustrations of the histology are very similar to the photograph from the section from this specimen. The microscopical picture is very different from that presented by the synovial villi so frequently seen in rheumatoid joints (*see Figs.* 159, 168). It is possible that the condition was caused by irritation of tuberculous toxins. There was no certain microscopical evidence of tubercle in the section examined,† nor could the affection be regarded as any recognized form of tuberculous disease of the joints. On the other hand, the involvement of a number of joints within a reasonable period shows a close resemblance to rheumatoid arthritis.

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* I am indebted to Dr. Basil Hood for the later history of this case.

† This statement is made with a little hesitation. The slides were submitted to an experienced pathologist who decided unhesitatingly against tubercle. The narrow tracts of (? endothelioid) cells occurring in the scattered masses of inflammatory cells must not be overlooked. Watson Cheyne considers endothelioid cells scattered irregularly or running in broad tracts through inflammatory tissue to be one of the two characteristic forms of tuberculous inflammation. Bacilli are found in and amongst them. (*Inflammatory and Toxic Diseases of Bone*, p. 57.) The tracts in the section were carefully searched for bacilli, and in vain (R. C. S.).

(To be continued.)

TUBERCULOUS HYPERPLASIA OF THE LARGE INTESTINE (HUMAN TUBERCLE BACILLUS).

A REPORT OF A CASE WITH UNUSUAL FEATURES.

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AND W. T. MUNRO,

MEDICAL SUPERINTENDENT, GLENLOMOND SANATORIUM.

IN the March number of the *Edinburgh Medical Journal,* 1931, we gave an account of this interesting condition (*Fig.* 175). We discussed at some length the pathology of the condition, reviewed the symptomatology and diagnosis, and were able to determine the type of tubercle bacillus, whether human or bovine, in the more recent cases coming under our observation. Since that paper was written another case worthy of comment has come under our care. The first point of interest in this case is that an elder sister was operated on in the Edinburgh Royal Infirmary for a similar although much less extensive lesion. In searching through the literature, and reviewing the histories of all the published cases which we could obtain, we have failed to find any instance of familial disease of this nature. Possibly this is not surprising when the comparative infrequency of the condition is considered. In all the previous cases in which we have been able to isolate the infecting virus a bovine type tubercle bacillus has been found ; and the bovine type is less likely to be responsible for familial disease.

Pulmonary tuberculosis due to the human type bacillus is notoriously a familial disease, but no familial instance of two cases of pulmonary tuberculosis due to the bovine type has come to our knowledge. Possibly some explanation of the familial incidence in this case can be given when it is said at once that the virus in this case was proved to be a human type tubercle bacillus ; but why should the disease appear in the two sisters as a tuberculous hyperplasia of bowel ?

CASE REPORTS.

A brief account will be given of *Case* 1. We are much indebted to Mr. D. P. D. Wilkie. Professor of Surgery, Edinburgh, who has allowed us to make full use of the clinical report from his wards.

Case 1.—S. W.. female, age 14 years, admitted on Nov. 11, 1925. Complaint—pain and borborygmi over the abdomen for five years, and latterly pain and some vomiting. Previous history, tuberculous glands excised from the neck in 1914. Patient was then 3 years of age, and this operation was done in the Cottage Hospital, Dunfermline : when seen in November, 1925, patient was thin, abdomen showed no peristalsis, but examination by barium meal showed retention of the meal in the lower ileum. Laparotomy—the lower six inches of the ileum were hypertrophied

and were resected and an end-to-end anastomosis was made. The histological examination showed the condition to be tuberculous. The patient made a perfect recovery. She is now in domestic service in Kinross and is very fit.

Case 2.—J. W., female, age 16 years. Glenlomond and Royal Infirmary, Dundee. The patient was in Glenlomond Sanatorium from December, 1928, to June, 1929. She then showed small glands in the neck and had complained of pain in the abdomen. Nothing much was noted on examination of the abdomen and

Fig. 175.—Hyperplasia of the large intestine. Bowel from ileum to splenic flexure. Note rugose formation of epithelium and thickness of gut on cross-section.

she went home apparently well. She remained well till January, 1931, when she began to complain of pain in the abdomen, some constipation or diarrhœa, at times vomiting, and loss of weight. Her attacks of pain had been constant to the right side of the abdomen about the level of the umbilicus. On admission she was very thin and her colour was poor with rather an earthy tinge. The amount of subcutaneous fat over the abdomen was fairly good. There were scars of old operative interference over the right forearm. She had at 6 years of age in the Sick Children's Hospital, Edinburgh, a subperiosteal resection of the right radius with a bone-graft. This was done for tuberculous disease.

On Examination.—*Abdomen*—no peristalsis noted. There was tenderness down the border of the right rectus almost into the right iliac fossa with the maximum tenderness just about the level of the umbilicus. There was the sensation of the presence of an elongated swelling running vertically in the position of the ascending colon. No glands were palpable. *Cervical area*—small glands could be felt in the posterior triangles. *Chest*—no sign of active disease made out, but there was prolonged expiration at the left apex. *Systemic disturbances*—the pulse-rate was 90 to 100 at rest, and the evening temperature up to 99·4°. The diagnosis appeared to be one of tuberculous hyperplasia of the cæcum and ascending colon.

DIAGNOSIS.—The history of increasing pain in the abdomen, and constipation over a number of months, pointed to some form of chronic obstruction. The patient was very thin, and there was the feeling of a tumour-like mass over the region of the cæcum. A barium meal was given. No abnormality was noted in the stomach itself. At five hours the meal was traversing the ileocæcal valve and the head of
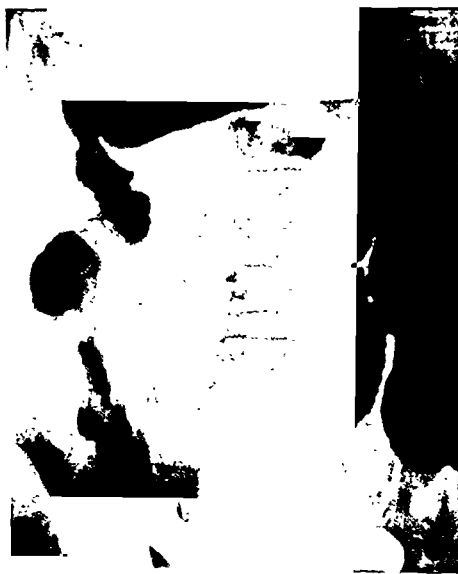
FIG. 176.—*Case 2.* Barium enema showing filling defects in ascending colon.
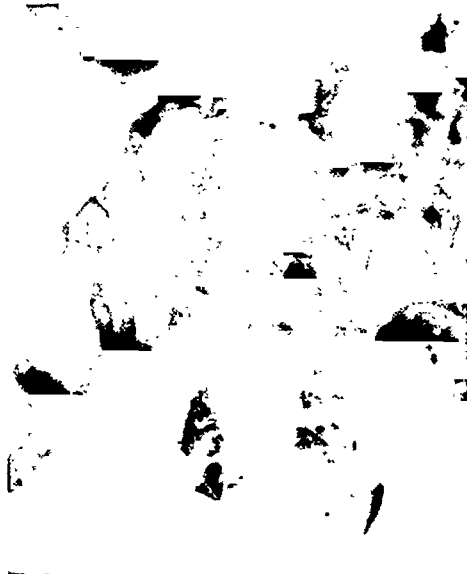
FIG. 177.—*Case 2.* Barium enema seven and a half hours after injection.

the column had reached the descending colon. The whole cæcum and ascending colon showed marked abnormality. The cæcum was drawn up. The ileocæcal loop was drawn out almost to a straight line. The cæcum was very narrow, the ascending colon also narrow, but there appeared some pouching as if there were areas of normal bowel in the ascending colon. Normal sacculation began at the hepatic flexure.

At a later date a barium enema was administered (*Figs.* 176, 177). This passed easily round to the cæcum and partly through the ileocæcal valve, confirming and showing more clearly the narrowed and irregular lumen of the ascending colon and cæcum. The limits of the irregularity seemed very sharply determined, for normal sacculation was seen at the hepatic flexure. A diagnosis of tuberculous hyperplasia involving the cæcum and ascending colon was confidently made.

OPERATION.—The patient was removed on Aug. 4, 1931, to the Royal Infirmary, Dundee, to the care of one of us (J. A.). The provisional diagnosis was corroborated, and on opening the abdomen by a right paramedian incision was fully confirmed.

The lower third of the ileum was found to show about twenty annular constrictions. These were healed tuberculous ulcerations which narrowed the lumen of the bowel considerably, but there was no thickening.

The cæcum and ascending colon were noted as almost solid in texture, and were stripped forwards and resected. The ileum was implanted into the lateral wall of normal transverse colon, while the proximal end of the transverse colon was invaginated and closed. The cavity was covered with peritoneum and the abdomen closed. The patient made an uninterrupted recovery and was discharged to the Sanatorium on Sept. 22. She had a somewhat irregular temperature but the bowel moved normally and she had no pain. To date (March 15, 1932) she has improved and is now gaining weight, while her systemic disturbance has almost ceased.

PATHOLOGY.—The cæcum and ascending colon were resected, and the disease ended abruptly at the hepatic flexure. The specimen showed a curious irregularity. There were two pouches of bowel about the size of half-a-crown which appeared normal, felt normal, and were quite pliable. Apart from that, the gut felt thick and inelastic. The appendix was embedded in fat and firmly fixed on to the cæcum, its walls were much thickened, and a thin serous fluid was present in its tip. The bowel felt turgid, mainly on account of the mass of mesenteric fat and glands drawn round it. The glands appeared almost to hang on to the wall of the gut. On opening the bowel there was about half an inch of fat around it, but the bowel wall itself was much less thickened than is usual in this type of lesion, a quarter of an inch being the greatest thickness encountered. The mucous membrane presented a curious rugose formation with many warty projections, much ulceration, and some thickening. Apart from the two areas where the mucous membrane seemed normal, the appearance of sacculation on the films, the bowel was simply a cylinder.

HISTOLOGY.—Professor Cappell reports : " The condition is frankly tuberculous, with ulceration and destruction of the mucosa in places, associated with massive round-celled infiltration. Tubercle follicles with caseation are present in the mucosa and submucosa, the latter being much thickened and fibrosed. Secondary infection of the ulcerated surfaces has, of course, occurred, and the surviving mucosa is slightly hypertrophic. The glands of the ileocæcal angle are slightly enlarged but show no caseation ; microscopically they are the seat of chronic tubercle follicles in small numbers. Tubercle bacilli have not been demonstrated in the sections ; this is hardly surprising in view of the chronic nature of the lesions."

BACTERIOLOGY.—No tubercle bacilli were found on examination of a crushed gland, and no gland had any caseous appearance ; but guinea-pigs inoculated with crushed gland, and with crushed bowel wall, all became tuberculous. The isolation of the infecting virus was effected at Glenlomond, and also by Dr. A. Stanley Griffith, Cambridge, to whom were sent some small enlarged glands and a piece of rugose ulcerated bowel wall. The lesions in the guinea-pigs were not marked, but glands and spleen were found infected when the animals were killed. Growths of tubercle bacilli were obtained both at Glenlomond and at Cambridge, and these proved to be eugonic, growing well on glycerinated egg and producing large, heaped-up, wrinkled growths. Rabbits were inoculated with the virus to make sure that there was no bovine strain present. The appearances of the rabbits with minimal lesions were those commonly seen after inoculation with a human type tubercle bacillus.

Dr. Griffith was satisfied, as were we at Glenlomond, that the virus was an ordinary human type tubercle bacillus.

COMMENTARY.

There are some features in relation to this case which are unusual :—

1. The first one—the familial incidence of a somewhat similar condition —has already been commented on. Neither patient appeared to show any active pulmonary tuberculosis. The fact that the infecting virus was found to be a human type tubercle bacillus is of first importance. One would have expected to find the bovine type in these cases, but from our experience the

familial incidence is against its being bovine, and the two sisters came to light as suffering from hyperplastic tuberculous disease of the bowel at much the same age.

2. The second point of interest in these two cases is that there were evidences of other tuberculous lesions in both. While one must always look for other lesions due to tuberculosis, those who have had experience of this intestinal condition have commented on the fact that other lesions have not been found. Indeed, the common experience is that this intestinal hyperplasia is the sole tuberculous manifestation, and pulmonary lesions are never found. But both the cases here have shown tuberculous cervical adenitis. Indeed, Case 1 had an excision of tuberculous cervical glands at three years of age, and Case 2 had a resection of the right radius at six years of age. The presence of cervical glandular lesions in both is of much interest, and one would have expected a spread towards the lungs in both cases, but Case 2 is the only one where some slight spread seems to have taken place.

3. The third noteworthy feature was the finding of so many healed tuberculous ulcers in the ileum in Case 2. This raises the question of route of spread of the disease in this patient. Did the small bowel and the large bowel become infected at the same time, and how did the bowel become infected ? Was such infection primary or secondary to a one-time active pulmonary lesion ? Was there a familial source of infection ? All these questions are difficult to answer. We have not been able to find an open case of pulmonary tuberculosis in the family, but the father is now in New Zealand, and it is stated he is not a fit subject, and may have been the infecting agent of his two daughters. We must remember that the child is a 'ground' animal, and human type tubercle bacilli carelessly deposited could readily be ingested by the 'ground' animal. Probably the healed ulcers in the ileum are not ulcers due to tubercle bacilli swallowed from a pulmonary lesion. There is not sufficient evidence of even old pulmonary disease to have allowed of this possibility. Primary infection of intestine can occur in the child and such lesions do heal and leave contracted areas. It is of course known that tubercle bacilli can pass through a mucous membrane without destruction of tissue, but primary ulceration in the child is common enough. We are inclined to the view that the starting-points of the lesions in small and large bowel were by the same infection. The lesions in the small intestine were healed, and in the large intestine proliferative, and this very proliferation is the evidence of healing, and at the same time the cause of the distressing symptoms.

4. The fourth unusual feature is the fact that the extent of the thickness of bowel wall is not as marked as is commonly the case in this condition, which may be in part due to the type of organism. While there is no proof —and indeed little to suggest—that the bovine type is less virulent than the human type to the human subject, yet it may be that the type of lesion produced by the human type is more a destructive than a proliferative one. At any rate, in those cases examined by us where the thickening of bowel wall was marked, the virus proved to be bovine type. Further observation on this subject is desirable. The question naturally also presents itself why this peculiar type of hyperplasia should arise at all. It has been suggested

that there may be some antecedent disease in the large intestine, but this seems to be quite unnecessary to explain the causation. The disease appears to us to be one due to lymphatic obstruction.

The lymphatic plexus of the large intestine is much less developed than that of the small intestine; and the drainage through the paracolic glands appears to give a lymph network which is much more easily obstructed than the lymph drainage of the small gut.

Mesenteric glands are very numerous and all of them at one time will not be blocked, and new lymphatic channels can readily be formed in the small bowel mesentery; but the case is different with the large intestine, where blockage of the few paracolic glands will result in lymphatic stasis and subsequently a thickening of bowel wall, with possibly a retrograde lymphatic flow and later some organization of the affected tissues.

In the case described all the paracolic glands were not noticeably enlarged, and the lymphatic obstruction was not complete. The abrupt end to the extent of the disease in these cases appears to be entirely dependent on the extent of lymphatic obstruction.

SUMMARY.

1. A case of tuberculous hyperplasia is described.

2. The diagnosis was confirmed by X-ray examination after a barium meal.

3. The infecting virus proved to be a human type bacillus.

4. Certain features unusual in this condition, as well as the possible causation of the hyperplasia itself, are considered.

We wish to express our indebtedness to Professor Cappell for his investigation and report on the tissues, and to Dr. A. Stanley Griffith for his confirmation of the type of bacillus.

A METHOD FOR THE PRODUCTION OF INCREASED COMPRESSION STRENGTH OF BONE: AN EXPERIMENTAL STUDY (PRELIMINARY REPORT).*

By DUDLEY ROSS,

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DR. EDWARD ARCHIBALD, of the Royal Victoria Hospital, Montreal, in 1924, proceeding upon the assumptions that in cases of fragilitas ossium (1) union nearly always occurs and therefore calcium metabolism is not at fault, and that (2) succeeding fractures rarely occur at the site of former ones and therefore bony union has strengthened the bone, carried out in one such patient an operation similar to that of Barth's,[1] although unaware of Barth's work. The latter in 1908 had removed longitudinal sections from the long bones in a case of juvenile osteomalacia, ground these sections into dust, and replaced the dust in the space from which the bone had been removed. In his report of the case he states that no further fractures occurred. Dr. Archibald's operation differed only in that the removed bone section was rongeured into small fragments instead of being ground into dust. These sections were placed in the medullary canal and the periosteum sutured over them. By this procedure he hoped that the bones would be strengthened, and the tendency toward repeated fractures lessened.

It is true that the fact that subsequent fractures do not occur at the site of previous ones is reported in the literature in several instances where cases were observed over a long period, but the reporters making these statements do not say how they arrived at this conclusion. As a matter of fact, the disease being one of childhood, it is difficult to state, on account of the growth in length of the bones, exactly where fresh fractures occur in respect of the site of a previous fracture. At two subsequent operations in Dr. Archibald's case, performed in 1928 on account of the occurrence of fresh fractures, small metal markers were inserted at the extremities of the opening left by the removal of the bony segment. In 1929 this patient suffered recurring fractures of both bones between the markers. A further attempt to increase bone strength was then made by doing the same operation on the opposite side to that of the first operation, in this case on the tibia, with the result that it gradually became clear that some better means of increasing bone strength had to be found if these cases of fragilitas ossium were to be helped, and the writer, at the suggestion of Dr. Archibald, began in 1929 an experimental investigation along these lines.

It was decided first to investigate experimentally the effect of the above operation on the compression strength of long bones of dogs, and on consulting with Dean Ernest Brown, of the Science Department, McGill University, the following method was evolved. After operation the bones were tested at various intervals in the testing laboratory of McGill University, and for this purpose a small Olsen testing machine of 10,000 lb. capacity was used. This
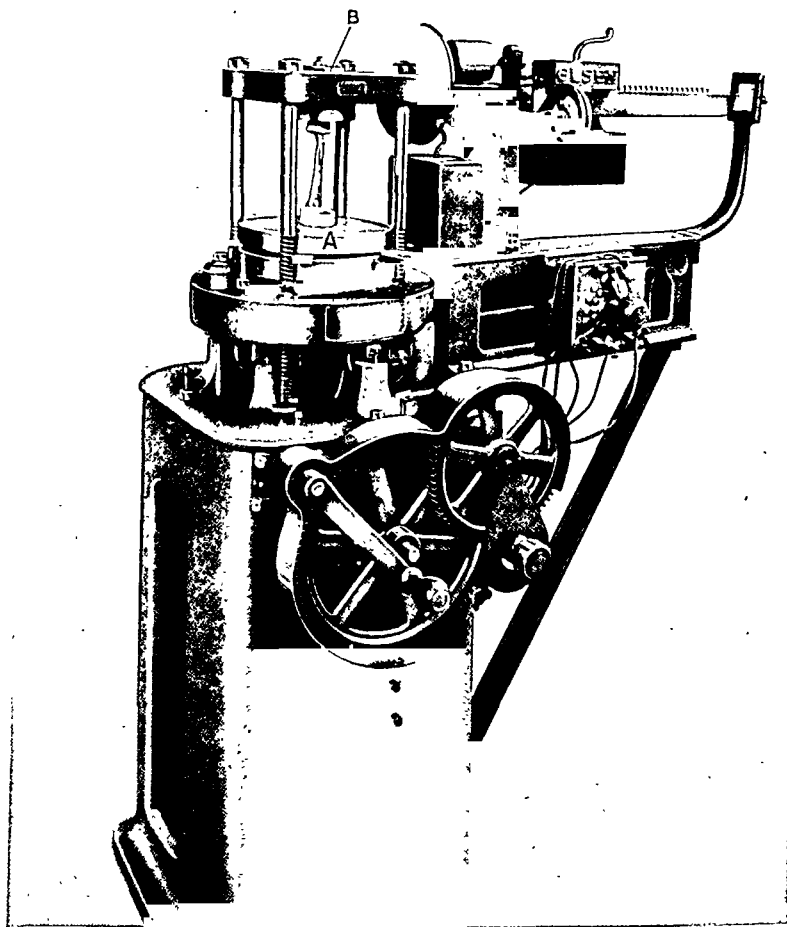
FIG. 178.—Bone mounted and in position for testing.

machine (*Fig.* 178) enables the bone to be loaded in compression, and a specimen arranged for testing is shown in the illustration. The ends of the bone were set in plaster-of-Paris contained in a ring of suitable diameter and depth, depending on the size of the specimen and the length of the bone operated upon. The plaster covered all the bone except that which had been operated upon. By this means it is believed that the load was distributed as uniformly as possible to the section under test, and that the results of such tests yield

reliable comparisons of the strength of the bone. The specimen shown in *Fig.* 178 is set up for the test, the two ends being embedded as described, and the bone being alined by eye so that its direction corresponded with that of the applied compression load. The specimen is set between a fixed base-plate A and a movable head B, the former corresponding to the platform of an ordinary weighing balance and being supported on a spherical joint which permits proper alinement. When a small initial load was carried and alinement secured, small wedges were inserted below A, to maintain the aline-ment. The load is applied by turning the gears shown in the foreground, thus rotating four vertical screws ; this rotation pulls the head of B down-ward, and subjects the specimen to compressive load, the amount of which is determined by the position of a balanced weight on a horizontal lever shown at the upper right corner of *Fig.* 178. The lever is maintained in a floating position throughout the test, and the load carried is weighed as in an ordinary balance. When the load reaches a value at which the specimen begins to fail, the lever no longer floats, and the dropping 'of the lever indicates the beginning of failure. These are the loads recorded in this paper, and while larger loads are required to break the specimens. the loads at incipient failure form a proper basis for comparison of the compressive strength of such specimens.

Young adult dogs were used in all cases. The right or left lower hind leg was prepared for operation and the opposite leg was used for control. A longitudinal incision was made from the knee to the ankle over the anterior surface of the tibia, and the periosteum of the latter was then exposed and incised, the incision extending from cancellous bone above to the cancellous bone below. It was then carefully elevated and retracted with a sharp periosteal retractor, and two parallel cuts were made with an Albee saw through the cortex, these cuts being joined at either end. The segment thus outlined was removed and rongeured into small fragments and placed in the medullary cavity, the periosteum was then closed by interrupted catgut sutures, and the skin by interrupted silk. Following the operation the animals were kept on ordinary laboratory diet and no attempt was made to splint the leg operated on. When any bony infection developed the animal was discarded. Those that lived and showed no bone infection were killed at various periods ranging from ten weeks to six months after the operation. Both tibiæ were removed and denuded of all soft tissue and were immediately mounted for testing.

EXPERIMENTS.

Dog 1.—Young male adult. Subperiosteal resection of the left tibia. No post-operative complications. Ten weeks after the operation the animal was killed and the tibia removed. Grossly the bone appeared similar to that of the other leg.

> Load capacity : Right tibia (control) 1130 lb.
> Left tibia   560 lb.

Dog 2.—Young female. Subperiosteal resection of the left tibia. Three months after the operation the dog was killed and both tibiæ tested.

> Load capacity : Right tibia (control) 866 lb.
> Left tibia   558 lb.

Dog 3.—Young female. Subperiosteal resection of the left tibia. Animal killed five months after the operation. Both tibiæ were removed and subjected to load capacity.

Load capacity : Right tibia (control) 430 lb.
Left tibia   439 lb.

Dog 456.—Young adult dog. Subperiosteal resection of the right tibia. In this instance a piece of lead was placed in the medullary cavity, the rongeured fragments being laid on top of it. Four and a half months after the operation the animal was killed.

Load capacity : Right tibia   148 lb.
Left tibia (control)   143 lb.

These experiments go to show that the operation as described fails to increase the strength of the bone operated upon, and also indicates that the return to normal strength does not occur until at least four months after the operation. In other experiments chemicals, such as iodine, acriflavine, and iodoform emulsion, were placed in the medullary cavity without producing any increased strength.

Leriche and Policard[2] report one case in which they inserted a fleshy granulation graft into a bony cavity in the tibia, with the result that the cavity was filled with new bone. It occurred to us, therefore, that as ossification can take place in muscle it would be possible, by implanting an isolated muscle-graft in the medullary cavity, to increase the amount of bone, and by so doing increase the compression strength in the long bones of experimental animals. The following series of experiments was carried out. An isolated strip of muscle was taken from the tibialis anticus and implanted along the whole length of the medullary cavity subsequent to removing a section of bone according to the technique already described. The removed section was rongeured and the chips applied on top of the muscle, and the cut edges of the periosteum then brought into apposition. Following the operation the dogs were given the usual laboratory diet. No splinting was applied. Fifteen animals were operated upon in this manner, but only four lived and were satisfactory for testing, the rest either dying of distemper or being discarded owing to infection. These four were tested at various periods following the operation, according to the technique outlined previously.

EXPERIMENTS.

Dog 462.—Young adult dog. Subperiosteal resection of the right tibia. No post-operative complications. On the 74th day the animal met with an accident and had to be killed. The tibiæ were cleaned and disarticulated at both ends. Grossly their external appearances were the same. The circumference of the right tibia, however, was increased over that of the left by $\frac{1}{8}$ in.

Load capacity : Right tibia   490 lb.
Left tibia (control)   385 lb.

Dog 50.—Young adult dog. Subperiosteal resection of the left tibia. No post-operative complications. Bones tested 107 days after the operation.

Load capacity : Right tibia (control) 138 lb.
Left tibia   290 lb.

Dog 33.—Young adult dog. Subperiosteal resection of the right tibia. No post-operative complications. Animal killed 147th day after operation.

Load capacity : Right tibia   1041 lb.
Left tibia (control)   836 lb.

Dog 34.—Young adult dog. Subperiosteal resection of the left tibia. The animal was killed 193 days after the operation.

Load capacity : Right tibia (control) 623 lb.
Left tibia 654 lb.
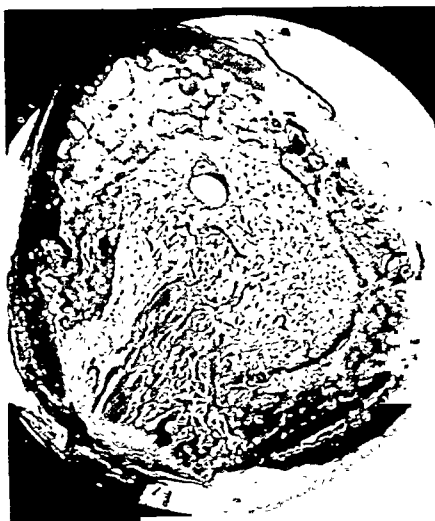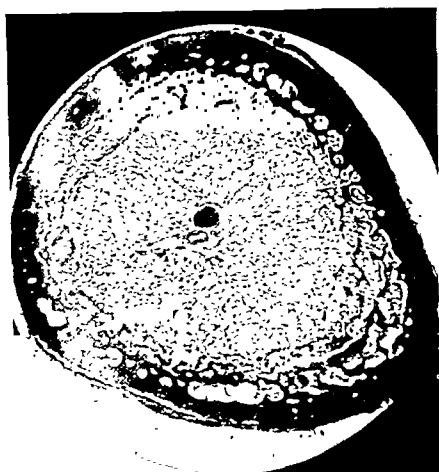
Figs. 179 and 180.—*Dog* 97. *Fig.* 179. Normal tibia. *Fig.* 180, Tibia 124 days after operation. The muscle has disappeared and has been replaced by fibrous tissue and new bone. The cortex is thickened and the circumference is visibly increased.

Figs. 181 and 182.—*Dog* 92. *Fig.* 181. Normal tibia. *Fig.* 182. Tibia 148 days after operation. The muscle has disappeared. No evidence of the operation. The cortex is thickened. The irregularity of the cortex is due to collapse of the section at the time of cutting.

While this last animal showed smaller increase than the other three, it was felt that this may have been due to the fact that a very small piece of muscle was used in the transplant, and that therefore there was not as great a production of bone as in the other animals. As a result of these experiments four animals were operated upon in order to investigate histologically the changes that occurred in the transplanted muscle and the bone. They were killed at various periods ranging from 74 to 260 days after the operation.

The histological sections were taken transversely through the middle of the shaft, hardened in Zenker's fluid, decalcified, and stained with hæmotoxylin and eosin. In none of the sections was there any evidence of muscle tissue, this latter apparently having undergone transformation into fibrous tissue, which was replaced by bone. The sections all showed thickening of the cortex and an increase in circumference.

Fig. 180 shows the reproduction of new bone, particularly at the site of the operation, and the replacement of the muscle by fibrous tissue and new bone. *Fig.* 179 is the normal tibia taken through the same site as *Fig.* 180. *Fig.* 182 shows the muscle to have entirely disappeared. The cortex of the tibia operated upon is thicker. as can be seen by comparing it with the normal (*Fig.* 181), and the site of operation is not visible as it is in *Fig.* 180. The appearance of the remainder of the sections corresponds to that of *Fig.* 182.

CONCLUSIONS.

1. Isolated muscle-grafts, inserted in the medullary cavity of the bone, disappear and are replaced by fibrous tissue and ultimately by new bone.

2. The increase in new bone increases the compression strength.

3. While it is generally assumed that the interposition of muscle between fractured bones prevents union, it would seem from the above experiments. that, providing the circulation to that particular section of muscle is cut off. union will occur in the usual time.

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SHORT NOTES OF RARE OR OBSCURE CASES

MACRO-GENITO-SOMIA.

By D. M. SUTHERLAND, MANCHESTER.

PRECOCIOUS sexual and bodily development (macro-genito-somia) may be associated with tumours of the suprarenal body, ovary, pineal gland, pituitary, or testis. Abnormal development associated with tumours of the testis is so rare that only two cases have been reported previously—one by Sacchi, of Genoa, in 1895, and the second by R. P. Rowlands in 1929 in the *Guy's Hospital Reports.* The rarity of the condition and the information which it may help to give on the internal secretion of the testis is the reason for the publication of the following case.

Fig. 183.—Photograph of patient.
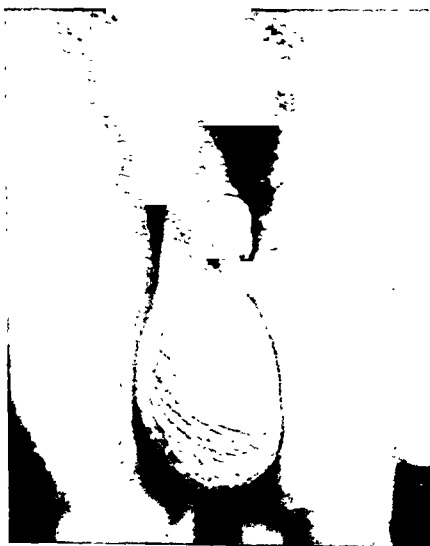
Fig. 184.—Showing dilated scrotal blood-vessels.

A boy, aged 11 years, was seen by me in the out-patients' clinic of the Royal Manchester Childrens' Hospital in December, 1931, suffering from an

enlargement of the right testis. The boy was small in stature, but the secondary sexual characteristics were fully developed (*Fig.* 183). He was very muscular, looked much older than his true age, and was very intelligent.

ON EXAMINATION.—The testis was found to be greatly enlarged and the scrotal veins were enormously dilated (*Fig.* 184). The testis was hard and sensitive to pressure. There was no obvious enlargement of the lumbar glands. The left testis was normal in size.

Radiological examination showed that all the epiphyses of the long bones had united. The pituitary fossa was not enlarged. The centre of ossification for the sternal end of the clavicle, which normally appears at 20 years of age and unites at 25, seemed to have almost united : it was possible to distinguish a thin line of separation between the epiphysis and the shaft.

FIG. 185.—Testis split along its anterior border.

I excised the testis in December, 1931 (*Fig.* 185). It measured about 4 in. in length and 3 in. in breadth. On naked-eye examination there did not appear to be any trace of normal testicular tissue or of the epididymis present. The testis was covered by an intact tunica vaginalis, not invaded by growth, but showing enormously dilated blood-vessels. The calibre of the spermatic artery was approximately that of the radial artery in an adult. The substance of the tumour was homogeneous reddish-brown in appearance and was partially divided by fibrous septa. There was one small area of hæmorrhage into the tumour.

MICROSCOPICAL EXAMINATION of the tumour showed that the cells were indistinguishable from the normal interstitial cells of the testis. They

exhibited the same staining reactions and contained lipoid material. *Fig.* 186, stained by Mallory's method, shows the homogeneous nature of the tumour under low-power magnification. *Fig.* 187, stained with hæmatoxylin and
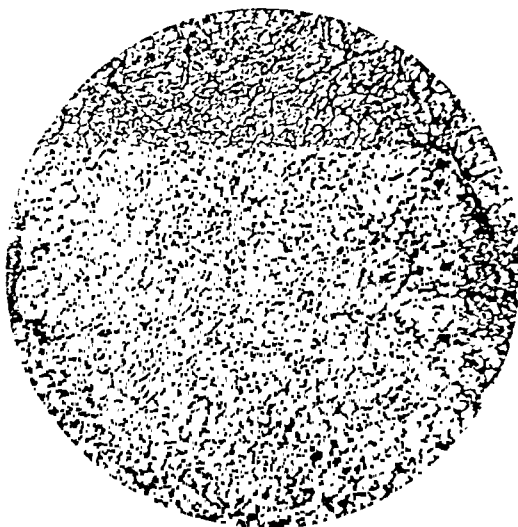
FIG. 186.—Section stained by Mallory's connective-tissue stain. (× 35.)

FIG. 187.—Section stained by hæmatoxylin and eosin. (× 250.)

eosin, shows the cells under high-power magnification: the clear spaces in the cells are the lipoid-bearing areas. There was no evidence microscopically of any of the cells of the tubules of the testis.

The history of the case suggests that the tumour was not malignant. The relatives of the boy stated that his premature development had been noticed by them four years previously, and said that the boy had been shaving since he was eight years old on account of his facial appearance.

SUMMARY.

A case is described in which overgrowth of the interstitial cells of the testis has caused premature normal development, and which appears to prove that the interstitial cells have an internal secretion influencing growth.

A CASE OF PROPERITONEAL HYDROCELE.

By JOHN McDONALD HOLMES,

THE DEPARTMENT OF SURGERY, LEEDS UNIVERSITY.

THE patient, a man aged 22, was admitted to hospital with a swelling of the left side of his scrotum of eighteen months' duration.   Six months after he first noticed the scrotal swelling an abdominal tumour appeared in the suprapubic region.   Both the scrotal and abdominal swellings enlarged painlessly until they reached the dimensions shown in the photographs (*Figs*. 188, 189).

ON EXAMINATION.—A bilocular cystic swelling was found in the scrotum. The left inguinal canal was distended, and a cystic tumour was found lying deeply in the suprapubic region extending up to the level of the umbilicus. This abdominal cyst communicated with the scrotal swelling through the inguinal canal, for a definite fluctuating impulse could be obtained in the scrotum on pressing the abdominal tumour, and again in the abdomen on pressing the scrotum.   The outline of the abdominal swelling did not entirely disappear when the patient made his abdominal muscles tense by raising his thorax from the bed.   The scrotal swelling could be transilluminated well in its upper part, and less well in the lower part.

The communication between the scrotal and abdominal swellings through the inguinal canal was rendered more evident by injecting 2 oz. of a saturated solution of sodium iodide into the scrotal cyst, allowing a little time to elapse in order that the solution might diffuse, and then taking a skiagram of the scrotum and abdomen.   The hydrocele was then partly evacuated by means of a trocar and cannula to prevent any symptoms which might have arisen from absorption of excess of sodium iodide.   Seven pints of pale yellow fluid containing suspended cholesterol crystals were drawn off, and approximately two pints were left in the cyst at operation.

OPERATION.—The hydrocele was subsequently removed by Mr. J. F. Dobson, through a left inguinal incision.   It was found to consist of three loculi, two in the scrotum, and one in the abdomen.   The abdominal loculus
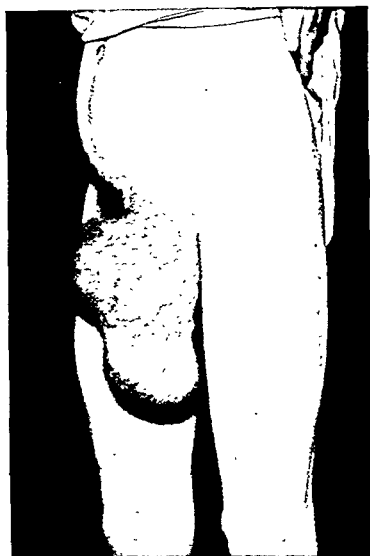
FIG. 188.—Semi-lateral view of the scrotum, showing the abdominal enlargement.
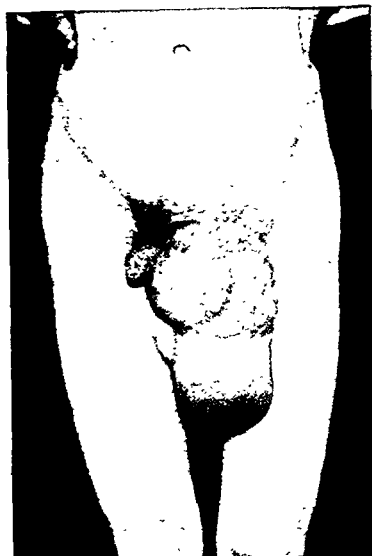
FIG. 189.—Anterior view of the hydrocele.

FIG. 190.—Skiagram after injecting the hydrocele with sodium iodide.

FIG. 191.—The entire hydrocele after removal, cut open to show the testis.

had evidently arisen from an unobliterated part of the funicular process lying within the inguinal canal. This unobliterated portion must have become distended with effusion after the scrotal sac had reached a moderate degree of enlargement, and the abdominal loculus must have been formed by its distension upwards and backwards through the internal inguinal ring. The abdominal loculus was found to be lying between the peritoneum and the anterior abdominal wall—that is, properitoneally. It was covered by a much thickened layer of subperitoneal connective tissue. The peritoneum was adherent to this layer over a small area at the upper end of the loculus, and was slightly torn in removing the hydrocele. The sac did not communicate with the general peritoneal cavity in any way. The obliterated funicular process was a short fibrous cord terminating in a small dimple as seen from inside the sac, and attached to the peritoneum at its upper end. The scrotal part of the hydrocele was very adherent to the scrotal tissues and cord, so the testis was included in the removal of the whole.

The small hole torn in the peritoneum was sutured, and the inguinal incision was closed as in a radical cure of an inguinal hernia ; first, by stitching the conjoined tendon to Poupart's ligament ; secondly, by joining the divided external oblique aponeurosis by means of imbricating sutures ; and lastly, by skin sutures.

The accompanying photographs (*Figs.* 188–191) · show the patient as admitted to hospital, the skiagram taken after injection with sodium iodide, and the hydrocele after removal.

I am indebted to Professor J. F. Dobson for his kind permission to publish this case.

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SPINAL TUMOUR SEEN ON DIRECT X-RAY EXAMINATION WITHOUT LIPIODOL.

By L. BATHE RAWLING,

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In my own experience, and from inquiries amongst my neurological colleagues, it is most exceptional for a spinal tumour to be seen on direct X-ray examination without the aid of lipiodol. Hence the reporting of this case.

A man, age 40, was admitted under my care with a diagnosis of spinal tumour. The signs and symptoms were suggestive of a tumour lying outside the cord at the level of the 4th and 5th dorsal vertebræ. An X-ray was taken, and herein lies the main interest of the case. The tumour was clearly visible in the middle line as an oval swelling, the size and shape of an olive, at the level suspected (*Fig.* 192). Cisternal lipiodol sank to the upper level of the tumour, sitting on its upper end like a man in his saddle (*Fig.* 193).

Froin's syndrome was present, and it was clear that there was a more or less complete block.

The tumour was exposed and removed. It lay in the middle line, attached by one or two strands to the inner aspect of the dura. the cord

FIG. 192.—Showing the olive-shaped tumour in the middle line at the level of the 4th and 5th dorsal vertebræ.

FIG. 193.—After cisternal injection of lipiodol.
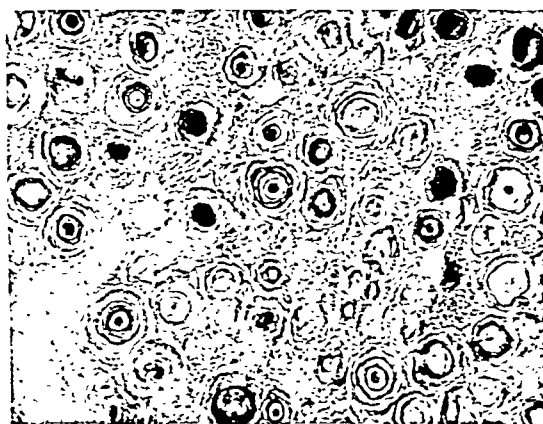
FIG. 194.—Microscopic structure of teratoma.

lying anterior and markedly compressed. No difficulty was experienced in its removal. and the patient is rapidly improving. The histology was that of a teratoma (*Fig.* 194).

REVIEWS AND NOTICES OF BOOKS.

Principles of Preoperative and Postoperative Treatment. By REGINALD ALEX CUTTING, M.D., C.M., M.A., Ph.D., Assistant Professor of Surgery, Louisiana State University Medical Centre. With a Foreword by RUDOLPH MATAS (New Orleans). Super royal 8vo. Pp. 812 + xx, with 76 illustrations. 1932. New York: Paul B. Hoeber, Inc. $10.00.

IT is very characteristic of present-day surgery that more and more attention is being devoted to the preparation of patients before operation and their management subsequently. This has meant the study of the physiological reactions of the body in health and disease and the application of such knowledge to surgical problems. How great the advance has been can be realized from the fact that nowadays a diabetic patient can be operated upon with scarcely any more risk than a normal patient. The complexion of the whole subject has been changed. This book differs from many such dealing with the same matter in that it attempts to expound the scientific basis for our present procedures. Thus the facts of water balance and dehydration are presented before a discussion of proctoclysis, infusion of saline, and hypodermoclysis.

A valuable chapter is that on disturbances of acid-base equilibrium, acidosis and alkalosis, in which the fundamentals of the subject, the properties of solutions, osmosis, pH, etc., are presented with great simplicity and clarity. Questions such as that of the administration of morphine, the control of gas pains, and vomiting are all carefully considered, and attempts are made to answer them in the light of known facts. The same method is employed all through, whether it be the management of urinary cases, thyrotoxic patients, head injuries, or post-operative lung complications.

We have nothing but praise for the book, which will be of interest to all surgeons, even those who have followed the literature of the subject; for the information is collected and the sources of it are given in an excellent bibliography appended to each chapter. There are a certain number of illustrations—as many as are necessary. The author's style is easy to read.

Surgical Errors and Safeguards. By MAX THOREK, M.D., Surgeon-in-Chief, the American Hospital, Chicago, etc. With a Foreword by ARTHUR DEAN BEVAN, M.D., Professor and Head of the Department of Surgery, Rush Medical College of the University of Chicago. Super royal 8vo. Pp. 696 + xiv, with 668 illustrations, many coloured. 1932. London: J. B. Lippincott Co. 45s. net.

IN his preface the author states: " While it is human to err, it is inhuman not to try, if possible, to protect those who entrust their lives into our hands from avoidable failures and danger. . . . Indeed, it is because of his own mistakes and the dangers which he himself has met that the author is filled with the keen desire to impress their possibility on others, so that they may benefit from his failures and disappointments." To achieve this laudable end the author has thoroughly searched the literature for recorded accidents and dangers and has, in addition, obtained the details of many unrecorded instances in the practice of colleagues and friends, while he relates unreservedly instances of his own personal shortcomings. As a result he has compiled what is probably the fullest account yet published of these surgical errors, and the only volume dealing with such that hails from America. In it are included errors in methods of examination, in diagnosis, in estimating the operative risks, in the pre- and post-operative care of patients, and in deciding upon the line of treatment, as well as the many errors and accidents that have actually happened during surgical operations. The lessons to be learned from these failures are appropriately

stressed, and many valuable practical points are presented from the author's great experience with a view to their prevention. The special difficulties and dangers associated with each region of the body are, in turn, fully considered—so fully, in fact, that it is not easy to conceive of many further mishaps than those here instanced that are ever likely to be met with. The volume will serve a most useful purpose if only it will tend to deter the undertaking of major operations by men relatively incompetent to perform them. While we can assure all progressive surgeons that they will find much of interest and profit in this volume, yet it is certainly one that should not be permitted to fall into lay hands.

Allgemeine und spezielle chirurgische Operationslehre. By Dr. MARTIN KIRSCHNER (Tübingen). Vol. II. Die Eingriffe in der Bauchhöhle. Royal 8vo. Pp. 574 + xii, with 395 illustrations. 1932. Berlin : Julius Springer. Paper covers, RM. 102 ; bound, RM. 108.

IT is difficult to describe this book in anything but terms of extravagant praise. The author, whom one would have thought exhausted by his share in editing the largest extant text-book of surgery, has now given proof of unabated vigour and skill. The present volume deals with abdominal surgery. The anatomy of the abdominal wall and all the methods of approaching the peritoneal cavity are clearly described, and then the general principles and the special details of all operations upon the stomach, intestines, biliary system, pancreas, and spleen follow and form the bulk of the book.

The outstanding feature of this work is the illustrations. We do not remember ever having seen anything so perfect in a surgical text-book. The remarkable feature about 395 coloured illustrations is the combination of exquisite artistic work both in drawing and reproduction with perfect accuracy of anatomical detail and clearness of diagrammatic presentation.

There is an ample recognition of mechanical aids to operative methods, and all sorts of contrivances from Murphy's button to Petz' sewing machine are included.

The description of operations is not confined to the formal procedures which can be carried out on the dead body, but include the real complications of disease— for example, the ulcers following gastro-enterostomy, the various types of paralytic ileus, and the pelvic and subphrenic abscesses which may follow appendicitis.

It is certainly a work that every medical library ought to possess, and it should afford a stimulus to British surgeons, artists, and publishers to produce a book of equal merit.

The Lame, the Halt, and the Blind. The Vital Rôle of Medicine in the History of Civilization. By HOWARD W. HAGGARD, M.D., Associate Professor of Applied Physiology, Yale University. Medium 8vo. Pp. 420 + xxvi, with 200 illustrations. 1932. London : William Heinemann (Medical Books) Ltd. 21s. net.

THIS book, written by the Associate Professor of Physiology in Yale University, belies its title. It is written for the American public and is a series of interesting essays under chapter headings the appropriateness of which is sometimes not apparent at first sight. Some of the essays are historical, some deal with old-world medical beliefs, whilst others are lay sermons upon the text that everyone should be thoroughly examined at least once a year with a view to the detection of the beginnings of disease. The advice may be good in theory, but in practice it would probably produce a race of neurasthenics. Amongst the subjects dealt with are the Black Death, rabies, the dancing mania, and tularæmia. There are some good short lives of Paracelsus and, as becomes a Professor of Physiology, of Dr. William Harvey and of William Beaumont, who elucidated the process of gastric digestion by his experiments on Alexis St. Martin.

The facts in the different essays are correct for the most part, but there is a good deal of embroidery which cannot be accepted as genuine. It was impossible, for instance, that Sir Walter Scott, who died in 1832, could have congratulated Sir James Y. Simpson, who received a baronetcy (not a knighthood) in 1866. Percivall Pott would certainly not have recognized himself as Dr. Pott, and Dr. Haggard's

republicanism has made him miss the point in his description of the King's Evil. Cures did take place as a result of, but not necessarily because of, the Royal Touch. The preliminaries were in reality a crude method of employing the open-air treatment of tuberculous glands.   The patients often came from the most remote parts of the British Isles.   The journey took weeks or even months and was made on foot or in slow wagons.   The dark overcrowded cottages were thus left behind ; the food was entirely different, and a careful selection of suitable cases was made by the royal physicians before patients were admitted to the Presence.   Then came a gorgeous ceremony followed by a second course of open-air treatment on the way home.

There are a few verbal slips which can easily be corrected, e.g., Cardimon for Cardamon ; Jacks for Jakes ; Melos for Melas.   It may be noted, too, that no witch was burnt in Scotland in 1882, though a picture is given of the alleged burning. It was not worth while to revive the folk etymology of Monk's Bane for antimonium, as the word was in use at least 400 years before the reputed Basil Valentine.

Take it all in all, the book is worth reading and will pass away some holiday hours.   It abounds in illustrations which are well reproduced and have been carefully chosen.

A Text-book of Neuro-Anatomy.   By ALBERT KUNTZ, Ph.D., M.D., Professor of Micro-Anatomy in St. Louis University School of Medicine. Medium 8vo. Pp. 539 +xii, with 197 illustrations.   1931.   Philadelphia : Lea & Febiger.   £50.50 net.

MANY text-books on neuro-anatomy are available, but this recent one by Professor Kuntz can be heartily welcomed as a valuable addition because of its distinctive qualities.   Whilst giving a very clear and concise account of the structure of the central nervous system, he has succeeded in advancing throughout the intimate relation which exists between form and function, and in correlating the " anatomical details with the fundamental structural plan of the vertebrate nervous system ".

The volume will be of service alike to students and to those who are qualified, since it includes a complete and up-to-date account of the structure of the brain and spinal cord, illuminated by knowledge derived from the study of phylogeny, ontogeny, and function.   Reference is made to recent work upon such subjects as the diencephalon, autonomic nervous system, cortical cyto-architecture, and neuroglia.

Although the book contains a vast amount of detailed information, it is attractively written and stimulates the reader by the able way in which the significance of the facts is introduced and the origin and modification of different parts are explained. The text is freely illustrated by nearly two hundred excellent diagrams and photographs of sections, many of which are original, and at the end of each chapter is to be found a list of monographs and papers which have reference to matters discussed.

Professor Kuntz is to be congratulated on the production of a book the perusal of which will prove profitable to anyone interested in the nervous system.

Orthopædic Surgery.   By WALTER MERCER, M.B., Ch.B., F.R.C.S.E., F.R.S.E., Assistant Surgeon, Royal Infirmary, Edinburgh, etc.   With a Foreword by JOHN FRASER, M.C., M.D., M.Ch., F.R.C.S.E., Regius Professor of Clinical Surgery in the University of Edinburgh.   Medium 8vo.   Pp. 695 + xii, with 371 illustrations.   1932.   London : Edward Arnold & Co.   32s. 6d. net.

THIS is a very complete text-book compressed into a comparatively small space. The necessary condensation does not always make for easy reading, but on the other hand it renders the book essentially suitable for the student, who will find it simple to look up and read a brief account of any particular clinical condition, including its pathology, differential diagnosis, and the principles of treatment. Actual methods, mechanical and operative, are described quite shortly, sufficient for the student, but in many parts insufficient for those who intend to carry them out themselves.   Any text-book of orthopædic surgery presents two essential difficulties : (1) That of arrangement, because it is almost impossible to classify the conditions that have to be dealt with entirely on either pathological or anatomical descriptions.   The author has not made any real attempt to overcome this difficulty.

Whereas affections of the spine, shoulder-joint, knee-joint, and foot each have separate chapters, it is necessary to consult the index to find affections of the hip or elbow. (2) That in many lines of orthopædic work there are still great variations between different schools of treatment, and a book written in Edinburgh is almost certain to be criticized in London or in Liverpool. As an illustration of this we may instance the author's account of the different opinions on the treatment of congenital dislocation of the hip, in which he states that one school of treatment believes the hip should be reduced as soon after the age of 2½ as possible, and, after quoting Galloway's view, says that a third group of workers would inaugurate treatment as early as possible. The last is surely the opinion that is almost universally held and taught.

An excellent feature of the book is the section given up to tumours of bone, which contains a careful and elaborate description based for the most part on recent American work. In spite of the great experience of these conditions which has been gained by the American Registry, it is still doubtful whether the nature of a tumour of bone can be diagnosed without a biopsy. Regarding the treatment of these tumours the usual pessimistic attitude about sarcomata has still to be taken, and no very definite line of treatment is put forward, although Kolodny's advice that the part should be treated by immobilization, radiation, and then amputation is mentioned.

The work is very well illustrated with radiographs, diagrams, and line drawings, and the arrangement of each section is sufficiently simple. There is a short list of references which does not attempt to be complete and which certainly leaves out a number that are really important. These references are arranged to correspond with the chapters of the book, and as a result it is impossible to find references to some of the subjects. To take the hip as an example, no references are given for coxa vara or osteo-arthritis of this joint.

The book should be a useful one for students reading either for a pass examination or for a higher standard, and also as a simple work of reference to the general practitioner.

Technique de l'Ostéosynthèse. By ROBERT DANIS, Professeur de Clinique chirurgicale à l'Université de Bruxelles. Royal 8vo. Pp. 162, with 149 illustrations. 1932. Paris : Masson et Cie. Fr. 55.

THIS monograph is devoted to a study of the technique of several types of osteo-synthesis which may be applied to the treatment of fractures. The use of rustless steel wire, inserted through drill holes and not encircling the fragments, is strongly advocated in preference to most other forms of internal fixation. Numerous instruments and an ingenious adjustable operating-table are described and well illustrated.

As an exposition of the technique of osteosynthesis—and in particular of the use of the wire suture—this work is interesting ; the consensus of surgical opinion, however, is that the operative procedures so enthusiastically defended by the author are very rarely necessary in the treatment of fractures at the present day.

Manipulative Surgery. By A. S. BLUNDELL BANKART, M.A., M.Ch. (Cantab.), F.R.C.S., Orthopædic Surgeon to the Middlesex Hospital, etc. Modern Surgical Monographs, edited by G. GORDON-TAYLOR, O.B.E., M.A., F.R.C.S. Demy 8vo. Pp. 150 + xii, with 21 illustrations. 1932. London : Constable & Co. Ltd. 7s. 6d. net.

MANIPULATIVE surgery is a valuable method of treatment, and it is kept prominently before the public largely because of the work of the unqualified practitioner and his methods of self-advertisement. This monograph, which deals briefly but very adequately with the subject, should be read not only by the specialist but by every practitioner of medicine. It is a personal statement of the views of the author on the uses and abuses of manipulation in surgery. It is dogmatic and is written with the simplicity and directness that would be expected from its author. In the introduction a brief account of anatomical, physiological, and pathological principles is first given, followed by a very short account of the methods and dangers of manipulations in general. The following sections deal in turn with the foot, the knee,

the hip, and other regions, the conditions amenable to treatment and the methods used being described in each case. There are some simple and clear photographs to illustrate methods. The book makes admirable reading and is full of valuable personal observations and opinions. The concise way in which important lessons are put may be illustrated by the opening sentence to the section on manipulation of the shoulder. " One of the most awkward situations in surgery arises when it has to be explained to a patient that his arm was broken while he was under an anæsthetic." There is a short chapter on bone-setting and osteopathy. On the latter Bankart is severe. " Osteopathy is a typical American money-making stunt." He advises inquirers to read the Bulletins of the A. T. Still Research Institute as a revelation of the way in which this subject can be written up in pseudo-scientific language for propaganda.

Allgemeine und spezielle Elektrochirurgie. By Dr. Med. HANS VON SEEMEN (München). Royal 8vo. Pp. 474 + xii, with 347 illustrations. 1932. Berlin : Julius Springer. Paper covers, RM. 62 ; bound, RM. 66.80.

IT would be idle to pretend that this book of 474 pages, interesting as it is, can be worth the 62 Reichsmarks (£4), which British readers (who of course pay Reparations excess) are asked to expend on it. Although 136 pages are devoted to apparatus, method of use, and the history of surgical diathermy in its various forms, it cannot be said that those who know nothing will receive simple instruction, or that those who are conversant with the electrotechnics of high-frequency apparatus will find anything very new.

The second or special part of the book is very lavishly illustrated, no doubt accounting for the high price. Many of the pictures illustrate surgical and pathological conditions as to which information would not be sought in a book on a technical method. To some extent this applies to the text also. At the same time, it is a demonstration that the cutting, coagulating, and hæmostatic currents can be applied in every field, from amputation of the limbs to excision of the upper jaw, from excision of the rectum to hæmangiomata.

The final section of the book, by Dr. Otto Schurch, deals with the combined employment of diathermy and irradiation, where there is, of course, a very wide field of utility. One of the advantages of diathermy undoubtedly is the diminution of sepsis in such situations as the mouth and rectum, when masses of growth are cleared out by the loop or by the flat coagulator. The authors say that after operation on rectal tumours, operable and inoperable, by diathermy, they have never seen either cellulitis or peritonitis.

The book, like all that come from Julius Springer, is beautifully produced, with very few printers' errors, and the translation of the well-known surgeon of St. Bartholomew's into the family of the bloody god Baal, may be only a tribute to his latter-day enthusiasm for the bloodless method of hæmostatic diathermy !

Die Formfehler und die plastischen Operationen der weiblichen Brust. By Dr. ERNA GLÄSMER, Frauenärztin in Heidelberg. Royal 8vo. Pp. 94, with 48 illustrations. 1930. Stuttgart : Ferdinand Enke. Paper covers, RM. 6.30 ; bound, RM. 7.65.

THOUGH plastic surgery has always featured prominently in the methods used by surgeons in this country in connection with certain deformities—for instance, in hare-lip—purely æsthetic surgery such as face-lifting and breast-moulding has had practically no position among the profession. What little has been practised has been carried on rather by the unorthodox practitioner. Foreign surgeons, on the other hand, have paid considerable attention to what is really one of the refinements of the art of surgery. For this reason British surgeons should welcome the appearance of the publication of this work by Dr. Erna Gläsmer, of Heidelberg. This small book of only 94 pages is an excellent exposition of all the most modern methods of breast-moulding and breast-lifting.

The operation in this country is practically confined to the correction of gross enlargements met with in parenchymatous hypertrophy, but apart from amputation

no text-book gives details of the procedure. The book contains not only details of the methods used to correct such gross deformity, but most of the methods employed to correct the ordinary pendulous breasts in women where sentiment or vanity—call it what you will—prompts this procedure. Practically the only method we have seen used in this country is the primitive one designed by von Lotsch, which sometimes gives good results. Needless to say, in order to attain anything like the precision required for success in these cases, great experience and constant practice are absolutely necessary. The results produced by the occasional performer are frequently anything but pleasing. The writer here can speak from experience. Besides these points the booklet deals with infantile breasts and the correction of depressed nipples. There are forty-eight excellent illustrations depicting different methods.

At the end of the book there is a list of 111 references to the literature of the subject, of which, however, only four are in French, one is in Spanish, and one is in English by an American and published in Germany. All the rest are German, which perhaps bears out what is said at the beginning of this criticism. The booklet is well worthy of the attention of those interested in the subject.

Die äusseren Abdominal-Hernien. By Erich Bumm (Berlin). Imperial 8vo. Pp. 331 + vii, with 235 illustrations. 1931. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 40.1; bound, RM. 42.50.

DR. Bumm states in his preface that the object of his book is to re-awaken and stimulate interest in the subject of hernia, which during the last ten years has fallen into disregard, although the cure of hernia is of the very greatest importance to so many human beings at the outset of their lives. This is certainly true; it may be doubted, however, whether Dr. Bumm's book will quite serve its intended purpose. It is a good account of the subject on conventional lines, but does not contain anything which arrests the attention or stimulates interest. The author's modesty seems to forbid the inclusion of any original observations, or even personal views as to the relative merits of the different forms of operation that are recognized. The really difficult problems, such as the treatment of direct inguinal hernia and of uncontrollable hernias in stout elderly people, are scarcely mentioned, so that the whole subject is left very much where it was ten years ago. The most important contribution to the problem of hernia made during the last decade has been Gallie's work on the proper use of fascial transplants, and this is not even mentioned in Dr. Bumm's book. The non-operative treatment of hernia is also completely ignored. The best feature of the work is the excellent series of illustrations. Many of these have simple colouring and are printed in the text, a method of illustration which is insufficiently used in this country. There is no index, either of subjects or of authorities—an unpardonable omission in a work which starts with the claim already quoted.

Division palatine: Anatomie—Chirurgie phonétique. By Victor Veau, Chirurgien de l'Hôpital des Enfants assistés, with the collaboration of Mme. S. Borel. Large 8vo. Pp. 568 + viii, with 786 illustrations. 1931. Paris: Masson et Cie. Fr. 140.

Traitement du Bec-de-lièvre unilatéral (Procédé du Dr. Veau). By Paul Plèssier, Ancien Interne des Hôpitaux de Paris. Large 8vo. Pp. 148 + iv, with 106 illustrations and 16 plates. 1931. Paris: Masson et Cie. Fr. 30.

Although the authorship of these two books is different, they are in reality complementary. The volume by Dr. Veau deals with cleft palate and its treatment, while the one appearing under the name of Dr. Plèssier, a former intern of Dr. Veau's, is essentially an account of his former chief's treatment of hare-lip. The name of Dr. Veau is well known in this country as an authority on these two important subjects, and the present publications are the outcome of experience gained in the treatment of over 500 cases of hare-lip and cleft palate.

The volume by Dr. Plèssier is perhaps the more formal of the two, and he reviews all the older methods of treatment of hare-lip and discusses the causes of failure.

All the technical details and steps in the operation as practised by Dr. Veau are described and illustrated.

Dr. Veau's volume on the treatment of cleft palate should be read by all who are interested therein. Not only is there a full discussion of the subject, but the details of some 500 case histories are appended and explained by a wealth of diagram and illustration. A valuable portion of the book is that contributed by Mme. Borel, who has been responsible for the after-training of patients operated on for cleft palate. There is in addition an analysis of 100 cases operated on between 1922 and 1926 and followed up. The author is to be congratulated on his presentation of a subject where accurate observation, careful surgical technique, re-education, and systematic follow-up are essential to any adequate exposition.

A Text-book of X-ray Therapeutics. By the late ROBERT KNOX, M.D., C.M. (Edin.), etc. Fourth edition, completed and edited by WALTER M. LEVITT, M.B. (Irel.), M.R.C.P. (Lond.), D.M.R.E. (Camb.), Medical Officer in Charge of the Radiotherapeutic Department, etc., St. Bartholomew's Hospital. Royal 8vo. Pp. 250 + xii, with 95 illustrations and 11 plates. 1932. London: A. and C. Black Ltd. 21s. net.

THE text-book of X-ray therapeutics by Robert Knox, completed and edited by Walter M. Levitt, is a worthy memorial to one of England's greatest radiologists. At the time of his death, Dr. Knox was engaged in the preparation of a new edition of his well-known book on radiotherapeutics. Owing to the lapse of time and the rapid advances in radiological methods of treatment, the task undertaken by Dr. Levitt must have presented many difficulties. These he has successfully overcome, and in the new edition, many chapters of which have been written entirely by Dr. Levitt, the whole subject of radiotherapy is fully dealt with in all its aspects.

After a brief introduction, the opening chapters deal with the effects of X rays on tissues, the physics of X-ray therapy, including X-ray measurements and apparatus, and with the principles of X-ray therapeutic technique. The physics are not too advanced for the average medical student, for which he will no doubt be thankful, and the principles governing the use of X rays in the treatment of disease are clearly and logically laid down.

Other chapters deal with the treatment of malignant disease and with diseases which affect the various anatomical systems. As an appendix there is a paper by the late Dr. Knox on " The biological factor as a guide to dosage and its influence upon the developments of technique ". A useful bibliography and an excellent index are provided. The illustrations are well chosen, and the publishers are to be congratulated on their part of the work. It is without doubt the best text-book on this subject which so far has been published in this country. It will be invaluable to students preparing for the Diploma in Radiology and to all radiologists who are practising this branch of the subject.

Primary Carcinoma of the Lung: Bronchiogenic Cancer: A Clinical and Pathological Study. By B. M. FRIED, M.D., Peter Bent Brigham Hospital, Boston, Mass. Royal 8vo. Pp. 247 + x, with 95 illustrations. 1932. London: Baillière, Tindall & Cox. 26s. 6d. net.

THE author states in the prefatory note that " clinician, pathologist and radiologist alike stress the frequent occurrence and significance " of primary carcinoma of the lung, and, were such necessary, this is the justification for the writing of this volume.

It is significant that the book is devoted entirely to the clinical and pathological aspects of the disease, and suggests that little or no attempt has been made to employ any form of active treatment other than X-ray therapy. Of this method of treatment in bronchiogenic carcinoma the author states it " may be dismissed by saying that its use has yielded no favourable results "—an opinion shared by the reviewer.

In the discussion on the incidence of pulmonary cancer the view is held that the increase of this disease is more apparent than real, and, further, that none of the suspected predisposing factors, such as influenza, tar inhalation, etc., can be said to be more than forms of pulmonary irritation.

The chapter on the histogenesis is of considerable interest, as the opinion is put forward that all primary pulmonary carcinoma is bronchiogenic in origin and results from excessive regeneration following irritation of the bronchial mucosa.

As only the basal cells are concerned in the process of regeneration, it is held that it is in these cells that the malignant process commences.   Thus the squamous- and basal-celled epitheliomas of the lung do not arise from metaplasia of the super- ficial ciliated columnar epithelium but from protoplasia of the undifferentiated basal cells.

A feature of special surgical significance is the large proportion of cerebral metastases.   In the group of forty-seven cases recorded in the monograph, no fewer than sixteen showed evidence of secondary deposits in the brain confirmed by operation or necropsy.   Thirteen of these were diagnosed as ' tumour of the brain suspect '.   Seven were operated upon as cerebral tumours without carcinoma of the lung being suspected or diagnosed until after operation.

This monograph shows evidence of wide study of the literature, careful analysis of the material supplied by a group of cases illustrating the protean manifestations of this disease, and, being clearly printed and well illustrated, can be heartily commended to all those who are interested in chest disease.

Die gesunde und kranke Wirbelsäule im Röntgenbild. By Prof. Dr. Georg Schmorl and Dr. med. Herbert Junghans. Large 8vo. Pp. 211 + xii, with 345 illustrations. 1932. Leipzig : Georg Thieme. Paper covers, M. 30 ; bound, M. 32.50.

This book is a study of normal and pathological anatomy of the spine in radiograms. It is therefore very important from the radiologist's point of view, as it is so necessary that he should know his anatomy and pathological anatomy thoroughly. The radiograms are all taken from pathological specimens, often from a sagittal section ; in many instances the photograph of the pathological specimen is placed on one side of the page and the radiogram on the other.   Thus, from the radio- graphic point of view, there are certain limitations, because a pathological specimen does not always give quite the same appearance as the living bone ; in particular this would be noticeable in the case illustrating angioma of the vertebeæ (p. 76). Also it is never possible in the living being to obtain quite so sharply defined a radio- gram when it is taken some distance from the vertebræ as one can get from a pathological specimen which can be laid directly on the film.   Nevertheless the radiograms are a very good indication of what one is liable actually to see in the living subject.   Most of the reproductions are either life-size or as near life-size as possible, many of them being three-quarters or four-fifths, and the amount of reduc- tion, if it occurs, is almost always indicated in the corner of the picture.   They are reproduced as positives, which is an advantage.

The work commences with a study of anatomical specimens of different parts of the vertebræ at various ages, and it shows clearly the development of the bodies of the vertebræ, but not so clearly that of the arches.   After the bodies, arches, and articulations are studied, the intravertebral discs, ligaments, and spaces between the vertebræ are described.   Congenital abnormalities are next treated in the same order, but the description of the various forms of spina bifida is reserved until later in the book.

The main portion of the book then deals with various diseases of the vertebræ, and in addition to common diseases many rare conditions are described with which fortunately the radiologist does not often come into contact.   We would welcome a somewhat clearer account of osteoporosis, but this condition is still apparently the subject of research.   In some cases the same may be said to apply to the description of some of the rarer conditions—for example, the Schmorl bodies first described by one of the authors.   The commoner diseases and traumata are well illustrated and are described at length.   The sacrum and the lumbo-sacral region are treated under a separate section from the rest of the diseases and injuries, though the reason for this is not apparent.   There is an extensive bibliography at the end of the work.

The whole of the monograph is very full and complete.   It is the more surprising,

therefore, that there is no index. This is, of course, a very great disadvantage in a book of reference, and in order to find some particular condition one may have to go through a large portion of the list of contents. There is no question, however, that notwithstanding this disadvantage it is a book which should be in the library of every radiologist and orthopædic surgeon as well as those general surgeons who are interested in this region.

Le Ascaridiosi chirurgiche. By Dott. ETTORE RUGGIERI. Medium 8vo. Pp. 256 + vi, with 29 illustrations. 1932. Lanciano : Giuseppe Carabba.

THIS is the first of a series of monographs to be published from the Surgical Clinic of the University of Parma. It runs to 250 pages, with a bibliography of 400 items, and sets forth all that is known about the *Ascaris lumbricoides* in its relation to man. There is an interesting account of the life-history of the worm, including the passage of the larva from the intestine to the liver, from the liver to the lung, and the arrival of the adult worm in the intestine via the bronchi, trachea, larynx, and pharynx. The experimental evidence of this cycle seems to be complete.

The pathological effects due to the worms are discussed under the heads of toxic and irritative action, mechanical, traumatic, infective, and what the author calls 'spoliative'—that is to say, in the diversion of foodstuffs. In the case of a single worm, this last is, of course, negligible ; but since more than one hundred worms may be found in the intestines of a child, it might have some importance. To English readers it may be a surprise that intestinal obstruction by worms is quite common and occurs in several ways. Diagnosis of obstruction due to worms is difficult, unless the subject is known to be infected or unless radiography be called in aid.

There are sections devoted to invasions of the biliary and pancreatic tracts by worms, and accounts of occasional invasion of nose, ear, bladder, uterus, and other parts.

It appears to be doubtful whether the ascaris can penetrate the normal intestinal wall, but there is no doubt at all that it makes its way through a wall damaged by ulceration, or between stitches of a sutured intestine.

The book is very well printed and contains many illustrations, including a number of very interesting radiograms. It should prove valuable to all who practice where helminthiasis is common.

Edemi chirurgici. By Dott. OSVALDO AMOROSI. Medium 8vo. Pp. 228 + vi, with 28 illustrations. 1932. Lanciano : Giuseppe Carabba.

THIS monograph on the surgical œdemas, from the University of Parma, hardly attains the standard set by the first of the series. It is, in fact, not more than a compilation. It adds little or nothing to existing knowledge, and it is difficult to discern for what public it is written.

The early chapters deal with the characters of œdematous fluids and with the various theories of pathogenesis, mentioning all the well-known names. There are short paragraphs about various 'causes', but, for example, one under the heading 'hæmoclasic crises' adds nothing to existing knowledge of anaphylaxis. There are, of course, differences in the œdema associated with glanders and with anthrax respectively, but they are incidental rather than basic.

There is an interesting paragraph about dense traumatic œdema following on repeated injury, but whether it is worth while treating it by Mantelli's adaptation of 'Lampson Handley's' silk-thread operation seems doubtful.

Elephantiasis comes in for fairly copious illustration, but very little is said about the treatment of extreme tropical cases. Throughout the book, in fact, treatment is perfunctory, but that is inherent in the study of what, after all, is only a single feature, common to almost every surgical malady.

There is a section on œdema associated with neuritis, which might well have been made the subject of a monograph, but the present work is an example of rather unnecessary bookmaking. The illustrations are poor.

Das Pollersche Verfahren zum Abformen an Lebenden und Toten sowie an Gegenständen. By ALPHONS POLLER. Edited by E. B. POLLER and E. FETSCHER. With a Foreword by Prof. Dr. C. VON ECONOMO (Vienna). Large 8vo. Pp. 216 + xii, with 129 illustrations. 1931. Berlin and Vienna : Urban & Schwarzenberg. Paper covers, RM. 12 ; bound, RM. 14.

THIS is a description of a method of taking casts of the living body or pathological specimens. The material for the mould is a substance called 'Negocoll', which becomes soft when warmed to a little above body temperature. When set it possesses great elasticity, so that overhanging and undercut moulds can be taken. This means few instead of many sections. Some very remarkable examples of casts are shown in photographs, such as clasped hands and faces which bear even the most minute skin creases. It is actually possible to paint Necocoll on to the surface of the cornea after cocainization. It is certainly a very important technical advance in the production of casts.

Some Radium Cases at the Middlesex Hospital: A Photographic Record. By A. CAMERON MACLEOD, M.B., B.S. (Lond.), F.R.C.S., Late Surgical Registrar, Middlesex Hospital. Large 8vo. Pp. 154 + viii, with 122 illustrations. 1931. London : John Murray. 7s. 6d. net.

THIS book is primarily a photographic record of patients suffering from various forms of malignant disease who have been treated with radium at the Middlesex Hospital. It does not pretend to be a statistical review, nor a serious text-book of radiotherapy. It consists of a number of photographs of patients before and after treatment with radium, and is intended to convince the sceptic that radium is of some value in the treatment of malignant disease.

Collected Papers of the Mayo Clinic and the Mayo Foundation. Edited by Mrs. MAUD H. MELLISH-WILSON, RICHARD M. HEWITT, M.A., M.D., and MILDRED A. FELKER, B.S. Vol. XXII, 1930. Medium 8vo. Pp. 1125, with 234 illustrations. 1931. Philadelphia and London : W. B. Saunders Co. 60s. net.

WE note that the steadily increasing output of the Mayo Clinic has necessitated a further addition to the editorial staff : the newcomer is fortunate to have the privilege of being trained by one who has done so much for the standardization of medical terms and editorial methods as has the lady known to most of us for so many years as Mrs. Mellish, now Mrs. Wilson. In the present volume the policy of the last few years has been extended. Of 482 papers available for publication only one-sixth are reprinted in full, 30 are abridged, 55 are extracted, and the rest are mentioned only by title.

Judd, Matson, and Mahorner contribute a valuable paper on ' Pancreatic Cysts ' with further records of 47 cases. These supplement Judd's paper of 1921 based on records of 41 cases from the Mayo Clinic, which soon became established as one of the classical papers of reference on the subject. The soundness of the conclusions which Judd arrived at in his original paper is shown by the fact that ten years further clinical experience has not demanded that any of these should be amended or reversed.

The contributions by James R. Learmonth, either alone or in association with others, on " The Sympathetic Nervous System in relation to Surgery of the Colon," will probably attract more attention than any other paper in this volume. They attempt to carry the surgery of the sympathetic nervous system a stage further than the work of Royle in an attack upon the post-ganglionic fibres rather than upon the ganglia themselves. It is much to be hoped that this writer will further continue this work both in the physiological and in the clinical field.

A paper by James C. Mason on " Extra-Uterine Pregnancy," quoted in abstract, is just one of those which particularly illustrates the value of this publication ; there can be no other writer who can claim to share with this author the advantage of possessing records of 471 cases observed in one Institution on which to base his conclusions.

Dr. Louis B. Wilson contributes an important résumé of the opportunities for the study of morbid anatomy in the United States and Canada which should be of value to anyone who is organizing a pathological department.

Reference has been made to only a few of the papers of surgical interest which are published in full and in abstract. From a study of the titles of papers mentioned only under this heading, it is clear that the importance and value of the output of the Mayo Clinic shows no signs of decreasing.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Intracranial Suppuration. By E. MILES ATKINSON, M.B., B.S.(Lond.), F.R.C.S., Surgeon-in-Charge of the Ear and Throat Department, Royal United Hospital, Bath. Modern Treatment Series. Crown 8vo. Pp. 127. 1932. London: Jonathan Cape Ltd. 5s. net.

Recent Advances in Anæsthesia and Analgesia. By C. LANGTON HEWER, M.B., B.S. (Lond.), Anæsthetist and Demonstrator in Anæsthetics, St. Bartholomew's Hospital, etc. Large post 8vo. Pp. 187 + viii, with 64 illustrations. 1932. London: J. & A. Churchill. 12s. 6d. net.

Kosmetische Operationen. By DR. ERNST EITNER (Vienna). Royal 8vo. Pp. 131 + vi, with 129 illustrations. 1932. Vienna: Julius Springer. Paper covers, RM. 18; bound, RM. 19.60.

Beitrage zur Mund- und Kieferchirurgie. By Prof. DR. G. AXHAUSEN (Berlin). Part 2 of Deutsche Zahnheilkunde, edited by OTTO WALKHOFF (Berlin). Large 8vo. Pp. 117, with 128 illustrations. 1932. Leipzig: Georg Thieme.

A Shorter Surgery. By R. J. McNEILL LOVE, M.B., B.S. (Lond.), F.R.C.S., Late Surgical First Assistant, London Hospital. Demy 8vo. Pp. 414 + viii, with 9 illustrations. 1932. London: H. K. Lewis & Co. Ltd. 16s. net.

Urology in Women. A Handbook of Urinary Diseases in the Female Sex. By E. CATHERINE LEWIS, M.S. (Lond.), F.R.C.S., Surgeon, Royal Free Hospital, London. Large 8vo. Pp. 76 + viii, with 21 plates and 4 illustrations in the text. 1932. London: Baillière, Tindall & Cox. 6s. net.

General Surgery. Edited by EVARTS A. GRAHAM, A.B., M.D., Professor of Surgery, Washington University School of Medicine. Practical Medicine Series. Crown 8vo. Pp. 804, with 173 illustrations in the text and 64 plates. 1932. Chicago: The Year Book Publishers. (London: H. K. Lewis & Co. Ltd.) £3.

Livre d'Or à l'Occasion du Jubilé de Vingt-cinq Ans d'Activité chirurgicale du Dr. Théodore Papayoannou. By Various Writers. Medium 8vo. Pp. 278 + xiv, with 110 illustrations. 1932. Naumburg-Saale: Lippert & Co.

The Melbourne Hospital Clinical Reports. Edited by S. O. COWEN, JULIAN SMITH, junr., et al. Vol. III. No. 1, June, 1932. Crown 4to. Pp. 58, illustrated. 1932. Melbourne: W. Ramsay (Surgical) Pty. Ltd. Annual subscription, 10s. 6d.; single numbers, 6s.

Minor Surgery of the Urinary Tract. By HERMON C. BUMPUS, junr., Ph.B., M.S. in Urology, F.A.C.S., Section on Urology, the Mayo Clinic. Medium 8vo. Pp. 124, with 57 illustrations. 1932. Philadelphia and London: W. B. Saunders Company. 15s. net.

Electrosurgery. By HOWARD A. KELLY, M.D., LL.D., F.A.C.S., and GRANT E. WARD, M.D., F.A.C.S. (Baltimore). Large 8vo. Pp. 305 + xxii, with 382 illustrations. 1932. Philadelphia and London: W. B. Saunders Company. 35s. net.

La Pratique chirurgicale illustrée. By VICTOR PAUCHET. Fasc. XVIII. Super royal 8vo. Pp. 264 + vi, with 189 illustrations. 1932. Paris: G. Doin et Cie. Fr. 65.

Surgery of the Chest. By GEORGE F. STRAUB, M.D., F.A.C.S. Royal 8vo. Pp. 475 + xviii, with 341 illustrations. 1932. London: Baillière, Tindall & Cox. 57s. net.

Verletzungen und Krankheiten der Kiefer. By Prof. Dr. GEORG PERTHES (Tübingen) and Prof. Dr. EDUARD BORCHERS (Aachen). Royal 8vo. Pp. 623 + xv, with 234 illustrations. 1932. Stuttgart: Ferdinand Enke. Paper covers, RM. 75; bound, RM. 78.

THE
BRITISH JOURNAL OF SURGERY

SOME BYGONE OPERATIONS IN SURGERY.

By Sir D'ARCY POWER K.B.E., London.

XI. THE REMOVAL OF A SEBACEOUS CYST FROM KING GEORGE IV.

Surgery had made much progress between 1737. when Ranby operated upon Queen Caroline of Anspach for a strangulated umbilical hernia. and 1821, when Astley Cooper removed a sebaceous cyst from the head of King George IV. But the progress was not sufficient to make a surgical procedure safe, and Astley Cooper's own account of his trepidation affords interesting reading for a surgeon of the present day. He says :—

" The King sent to Sir Everard Home, myself and Brodie to go to Windsor to see a tumour on the summit of his head. which annoyed him from its appearance. and was growing larger. When we saw it. it was tender, painful and somewhat inflamed ; and we thought it best to delay the operation. The King was much disappointed. but yielded to our advice.

" In the spring of the succeeding year. 1821, Sir Benjamin Bloomfield came for me to go to Brighton to see the King and I went down with Bloomfield. The King came into my room at one o'clock in the morning and said 'I am now ready to have it done. I wish you now to remove this thing from my head.' I said 'Sire—not for the world now—your life is too important to have so serious a thing done in a corner. Lady C—— S—— died of Erysipelas after such an operation and what would the world say if this were to be fatal ? No, too much depends upon your Majesty to suffer me at one o'clock in the morning in a retired part of the Pavilion. to perform an operation which however trifling in general, might by possibility be followed by fatal consequences.' He hastily said. 'This is the second time I have been disappointed.' 'Yes, Sire. I am sorry for it. but I should not choose to do it. unless Sir E. Home. Mr. Cline and Mr. Brodie were present. Mr. Cline. the world would say. would not advise anything without due consideration and from my long knowledge of him. too, I should like him to assist me and he would not object to bear a part of the onus under any circumstances.' He said. 'Well. I respect Cline and I dare say he respects me. although we do not set our horses together in politics.' I replied, 'Perhaps not. your Majesty. but

the best policy, I think, will be to have him.' 'I will have it done as soon as I come to town then.' 'Very well, Sire,' and on the following morning at eleven o'clock, I returned to London.

"The King came to town soon afterwards and I went to the levée and he said, 'How do you do, Cooper?—well, next Tuesday.' I called upon Lord Liverpool and requested him to persuade the King to let Home do the operation, as that was the usual etiquette, he being Serjeant Surgeon. Lord Liverpool said that it was very difficult to interfere respecting the choice of a medical man. I was very averse from doing it ; I had always been successful and I saw that the operation, if it were followed by erysipelas, would destroy all my happiness and blast my reputation. On the next day Home wrote to me that he should do the operation on Wednesday and I requested that Cline might be present to which Home consented. On the Wednesday we all met, Halford, Tierney, Home, Cline, Brodie and myself. Mr. Cline said 'Who is to do the operation?' I said, 'Sir Everard.' Soon afterwards Sir Henry Holland was called out of the room and almost immediately returning said to me, 'You are to do the operation.' ·

"I was thunderstruck and felt giddy at the idea of my fate hanging upon such an event. I said 'I have not come provided with instruments.' There was no time for parlance for the King directly entered the room and said 'Where am I to sit?' I replied 'here, Sire,' taking a chair to the window and begging an instrument of Home, I made an incision into the tumour and emptied it of its contents. Then I found it adhered strongly to the scalp and upon the side on which I stood which was about three-fourths of its size, I with difficulty detached it from the skin without cutting the skin itself. On that side on which Cline stood I begged him to detach it which he did but it took up a great deal of time on the whole. The edges of the wound were brought together and lint and plaister applied.

"The King bore the operation well, requested that there might be no hurry and when it was finished said 'What do you call the tumour?' I said 'A steatoma, Sire.' 'Then' said he 'I hope it will stay at home and not annoy me any more.'

"The King went on well until Saturday ; when he came in to us, he said 'I have not slept all night and I am damned bad this morning ; my head is sore all over.' I immediately thought erysipelas was coming on and that we should lose him. I called in the middle of the day at Carlton Palace and again in the evening and he was much the same.

"The next day when I went the King was on the sofa—his great toe was red with gout—and his head had lost its soreness and all its unpleasant feelings. From this time the wound healed in the most favourable manner.

"In a fortnight afterwards he said 'Lord Liverpool has promised to make you a Baronet but I will not suffer it, I shall do it myself.' I thanked him and said 'Since your Majesty is so kind let me say, if it be not entailed upon my nephew Astley whom I have adopted and educated it will lose much of its value.' He immediately said 'It shall be made out as you wish.'

"He afterwards in six months sent me a beautiful épergne for which he gave the plan himself and which cost him five hundred guineas and we continued the best friends."

Three days after the operation Astley Cooper was sent for hurriedly to see the King and returned smiling and saying that it was a false alarm and added " but pray tell me do you see anything particular about me ? for the King did not seem in good tune ; he looked very hard at me from head to

SIR ASTLEY PASTON COOPER, Bart.

Reproduced from the Painting by Sir Joshua Reynolds.

foot and I cannot understand why—do you see anything ?—Why I said I should have put on a white cravat and a clean shirt or at least have washed my hands before I waited on his Majesty. The fact is that Sir Astley had performed a slight operation just before he went to the Palace by which some

blood had stained the sleeve of his shirt, where it projected from the wrist and his hands were also not perfectly free from it. Mr. Cooper then looking at what I had pointed out to him said " God bless me. so I ought but I was not aware of it—the King. Sir, is very particular—he was lying on a couch under a canopy with a red turban on his head and looked displeased—and now I see the reason of it."   Dr. Badeley, who was then acting as Cooper's assistant, tells the story and Bransby Cooper reports it.

This account shows that there was a very real danger of the operation leading to some form of acute septic poisoning then summed up under the general term 'erysipelas'. It might have come from the surgeon because Astley Cooper considered any day wasted unless he had dissected for at least an hour, and this at a time when subjects were not injected and had often been obtained by resurrection methods. From the King because he was in poor health, very fat, and so plethoric that 130 oz. of blood had been taken from him to relieve an attack of suffocation from which he had suffered in the February of the preceding year.

Of the actors in the drama, Henry Cline, whose judgement was considered of such great importance, had been Astley Cooper's master at St. Thomas's Hospital. He was radical at a time when politics counted in everyday life. He was a devoted adherent of Horne Tooke, the agitator, and of John Thelwall, the reformer who used to cut the froth off a pot of porter and invoke a similar fate on all kings. Cline, too, was known to be in favour of the French Revolution. It was said on the other hand that his character was like that of Washington, for he devoted himself to what he considered to be to the advantage of his country and surrendered any distinction he might have attained when he had accomplished his object. He resigned his post of Surgeon to St. Thomas's Hospital in 1810, and in 1821 he was aged 71.

Astley Cooper, aged 53, was recognized by general consent as the greatest operating surgeon of his generation. He was Surgeon to Guy's Hospital and lectured at St. Thomas's, for the two hospitals were not separated until 1825.

Sir Everard Home, aged 65 in 1821, was the brother-in-law of John Hunter. He was Surgeon to St. George's Hospital and had been appointed Sergeant-Surgeon to the King in 1808. To salve his mortification at not being allowed to operate, his son, who was then a very young lieutenant in the Navy, was promoted out of his turn to the rank of Commander.

Benjamin Brodie was only at the beginning of his career, for he was not born until 1783. He had been house surgeon at St. George's Hospital under Home and had probably come with him to assist at the operation. It is evident, however, that his pleasant manners and gentle touch made a favourable impression upon the King, who afterwards consulted him frequently and appointed him Surgeon-in-Ordinary in 1828. He became Surgeon to St. George's Hospital in July, 1822, having already acted as Assistant Surgeon for fourteen years.

Sir Henry Halford was President of the College of Physicians from 1820 until 1844. He was a favourite with George III, who conferred a baronetcy upon him, and he attended George IV, William IV, and Queen Victoria. He is described as vain, cringing to superiors, and haughty to inferiors, but he was a good practical physician.

Sir Matthew Tierney was Physician-in-Ordinary to George IV. He was in practice at Brighton, and throughout his life was a strong supporter of vaccination. As a protégé of Lord Berkeley he had made Jenner's acquaintance when he was living at Berkeley Castle.

Lord Liverpool was Robert Banks Jenkinson (1770–1828), the second Earl of Liverpool, who was Prime Minister for fifteen years.

Sir Benjamin Bloomfield was the confidant of George IV and acted as his private secretary. He fell into disfavour in 1822, was sent as minister plenipotentiary to the Court of Stockholm, and was raised to the Irish peerage as Baron Bloomfield of Oakhampton and Tipperary in 1825. His social and musical attainments brought him to the notice of George IV, when he was living at Brighton doing duty with the 10th Hussars.

THE RELATIONSHIP OF THE STRUCTURE OF THE ENLARGED PROSTATE TO THE END-RESULTS OF PROSTATECTOMY.*

By E. W. RICHES,

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In assessing the prognosis and results of prostatectomy for enlarged prostate, attention has hitherto been confined mainly to two considerations, the personal factor of the patient and the type of operation performed. As regards the patient, the duration of symptoms and consequent degree of renal impairment appear to be of more importance than his actual age, and the presence of extra-urinary disturbances undoubtedly affects the final results in a series of cases. As regards the type of operation, discussion still continues on the relative merits of suprapubic, perineal, and perurethral prostatectomy, and of the open or closed operation in the first procedure. Many variations in after-treatment are also in use, and for each some benefit is claimed in reducing complications and improving results generally. One factor in prognosis which appears to have received insufficient consideration is the histology of the prostate removed. Cases of frank carcinoma, or those which prove to be malignant on microscopical examination, are notoriously difficult to remove and prone to recur ; but even if these are excluded it is found that some prostates are enucleated easily and others with difficulty, so as to necessitate dissection, and there is a general impression that the former are more satisfactory in their end-results than the latter.[1] The present investigation has been undertaken in order to establish, if possible, statistical evidence of a correlation between the type of prostate, the symptoms produced, and the incidence of complications and ultimate prognosis after prostatectomy. For this purpose all cases of prostatectomy at the Middlesex Hospital from 1924 to 1931 have been reviewed. They number 114, excluding those of malignant disease of the prostate, and include the results of nine different surgeons. All the cases were submitted to suprapubic prostatectomy, and this is the one common factor : the majority were carried out by Freyer's technique, but in some the open operation of Thomson-Walker was performed ; ligature of the vas was added in a few cases. The after-treatment given differed according to the practice of the individual surgeon. Despite these variations. the relative incidence of inflammatory complications was about the same

throughout the hospital, a fact which lends support to the view that in determining the subsequent course of the case there is some other factor concerned besides the technique employed in the operation and after-treatment.   All the prostates have been examined microscopically. and as a result a histological classification is suggested.

ANATOMY.

A brief description of the more salient features of the anatomy of the prostate is germane at this point.

The prostate consists of 50 to 60 glands. each opening into the urethra, with duct, ductules. and acini : 12 to 14 of these glands lie in a loose stroma of their own, which radiates out from the posterior aspect of the urethra to join the capsule of the gland ; these are called the *posterior lobe*—the 'true prostatic glands' of the Continental writers.   They form the whole of the posterior surface and the greater part of the lateral surfaces of the prostate. Their ducts, larger and longer than the others, converge to open around the orifice of the utriculus masculinus.

The remaining prostatic glands lie in the dense muscular tissue around the urethra, and are called the *peri-urethral glands* or central glands (Albarran and Motz[2]).   According to their situation they are divided into four groups or lobes as follows : Two *lateral* (14 to 16 in number), the ducts opening into the groove on either side of the crista urethralis ; *middle* (8 to 10), opening into the urethra above the utriculus masculinus ; and *anterior* (4 to 6), which open into the anterior wall of the urethra and are occasionally absent.

Albarran's glands, a few small glands opening into the posterior wall at the internal meatus, are often continuous below with the middle lobe.

While the lateral, middle, and anterior glands are continuous with each other, the posterior lobe is partially separated from them by the outermost layer of plain circular muscle, sometimes incomplete, which surrounds the urethral musculature and is continuous above with the vesical sphincter. The term 'peri-urethral glands' applies therefore to the lateral, middle, and anterior lobes, and the application of this name to small submucous crypts in the prostatic urethra is wrong and misleading.

PATHOLOGY.

Cases of non-malignant prostatic obstruction are clinically of three main types : (1) The typical 'enlarged prostate.' usually an easily enucleated tumour of some size ; (2) The small 'fibrous prostate'; and (3) The prostate in which the presence of calculi is the predominant feature.   Histologically there is some evidence that the two former are both examples of the same pathological condition—namely, benign hypertrophy—while the calculous prostate. unassociated with benign hypertrophy, is a separate entity.   The large fibrous prostate described by some writers is probably an intermediate form between the glandular and fibrous varieties.[3]

1. **Benign Hypertrophy** (*Adenoma of Prostate. Hypertrophic Prostatitis*).— A variety of names has been applied to this condition. the characteristic feature of which is the presence of multiple small areas of glandular tissue

in the prostate, which by their growth compress the surrounding substance, giving the appearance of an encapsulated tumour. A collection of these nodules lying in the muscular stroma around the urethra forms the mass which is commonly called an 'adenoma' of the prostate (*Fig.* 195).*
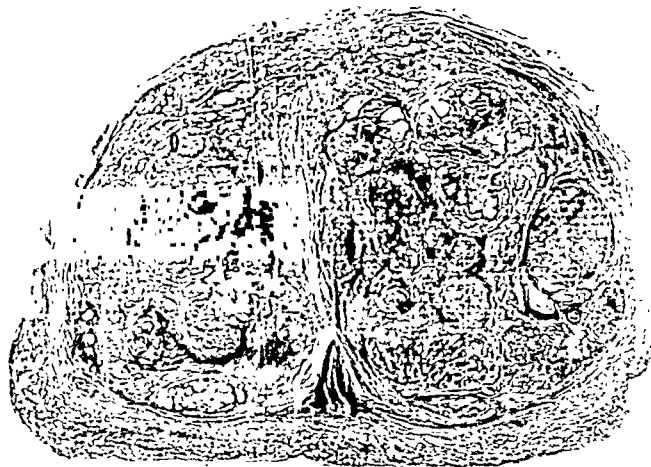
FIG. 195.—Section immediately above utriculus masculinus. Both lateral lobes are enlarged by many glandular nodules, some cystic, causing urethral deformity. Posterior lobe compressed. Age 80.

Site.—In 59 prostates removed at autopsy in which benign hypertrophy was present, the areas involved were as follows :—

	PER CENT	
Lateral lobes	90	
Middle lobe (associated with lateral)	34	
Middle lobe (alone)	6	(All early cases)
Anterior lobe	1	
Posterior lobe	6	(Associated in every case with lateral-lobe changes)

(Albarran's glands are included with the middle lobe.)

Both the anterior lobe, which consists of only a few small glands, and the posterior, which forms more than half the bulk of the glandular tissue, are seldom involved.

Histology.—The microscopic appearance is familiar, and shows small, dilated, or cystic acini lying in a varying amount of fibromuscular stroma. The epithelium is that of the normal prostate, or in several layers with papillary processes. The acini may be empty, contain secretion, desquamated cells, or corpora amylacea. The stroma is commonly compressed and atrophic. Rarely muscular tissue may appear to form the greater part of the tumour.

* All the illustrations are from autopsy specimens except *Fig.* 210, which is from an operation specimen.

particularly if a section is examined from that part of the adenoma adjacent to the anterior urethral wall, where muscular tissue is abundant in the normal prostate.

The earliest stages in the formation of the small nodules or 'spheroids' can be seen in autopsy specimens. The first sign of their appearance is a group of acini around which the neighbouring tissues appear distorted or compressed (*Fig.* 196). The majority of the prostatic glands in which these early changes have been seen were normal except for somewhat exaggerated. thin papillary processes. In others this papillary hyperplasia starts in cystic acini, and here the histological appearance is similar to that described in the

FIG. 196.—Nodule showing compression of surrounding tissues simulating encapsulation. From the middle lobe. Age 63.

breast by Cheatle.[4] Adjacent papillæ come into contact. fuse, and give the appearance of another acinus.

Jacoby[5] believed from histological evidence that these acini retained communication with the urethra. Herring and Lawrence[6] attempted to demonstrate this fact by the injection of fusible metal. This method entails the subsequent destruction of the prostate, and no histological evidence can therefore be obtained. The difficulty is obviated by the use of Prussian blue in gelatin injected per urethram on autopsy specimens, and sections can subsequently be cut. These show that where the proliferating area is small. dye will frequently enter the acini (*Fig.* 197). though not when definite encapsulation is present. In the latter case this probably due to the compression of the surrounding tissues and ducts.

Cysts in benign hypertrophy become increasingly frequent in the later decades (*see Figs.* 195. 199).

AGE	HISTOLOGICAL APPEARANCE OF ACINI IN 'SPHEROIDS'
40–49 } 50–59 }	.. Small acini. cysts rare.
60–69	.. Cysts in 40 per cent. In 5 per cent cysts predominated.
70 onwards	.. Cysts in 70 per cent. In 30 per cent cysts predominated.

The presence of cysts with incomplete septa and the fact that stages in their formation are frequently seen, such as the breaking down of walls between adjacent acini, suggest that cyst formation is the end-result of the epithelial proliferation, acini with desquamative changes representing a preliminary stage.

The presence of cystic acini was used by Ciechanowski[7] as evidence of duct stenosis with distension. Papin and Verliac[8] pointed out that while one part of the lining epithelium in a cyst might be flattened, in another high columnar cells might be present, and concluded that this appearance was
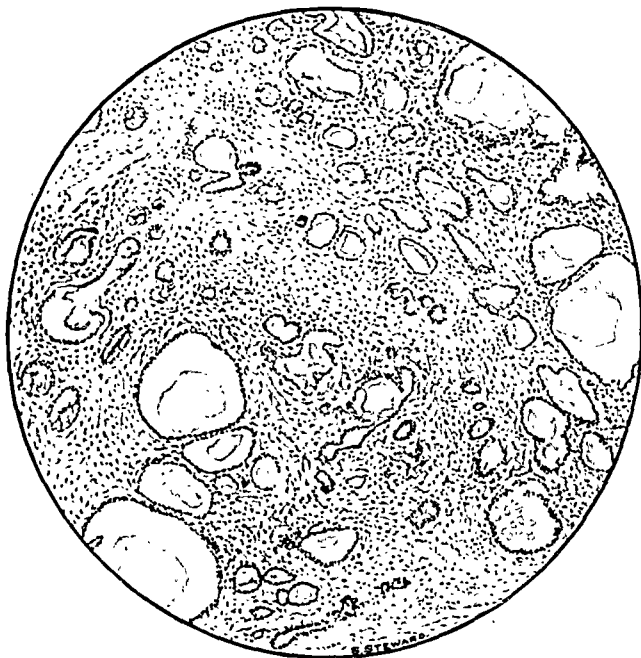
FIG. 197.—Dye injected from urethra into prostatic acini, in benign hypertrophy. Age 65.

not compatible with duct stenosis. Dye will enter these cystic acini with even greater ease than the small acini, and in the cases here examined no evidence of duct stenosis was found.

Posterior Lobe.—The changes found in the posterior lobe in cases of benign hypertrophy are usually described as those of 'compression atrophy'. That this is not the correct explanation is shown by the presence of similar atrophic changes in prostates without benign hypertrophy in men over 50.

BENIGN HYPERTROPHY IN LATERAL LOBES (Men over 50)	WITHOUT BENIGN HYPERTROPHY (Men over 50)
Atrophic acini present in posterior lobe in 77 per cent.	Atrophic acini present in posterior lobe in 70 per cent.
(In only 17 per cent of the above were the lateral lobes of sufficient size to have caused compression.)	

The changes seen in and around these atrophic acini are desquamation, fibrosis, and peri-acinar lymphocytosis. With the complete or partial shedding of the lining epithelium the corpora amylacea may act as foreign bodies and become surrounded by giant cells (*Fig.* 198). Cystic acini lined by only a trace of flattened cells are common, particularly on the lateral aspects of the prostate (*Fig.* 199). In 10 per cent of cases these cysts show the same papillary projections as those found in benign hypertrophy in the lateral lobes.

Young[9] illustrates a case of an encapsulated adenoma in the posterior lobe. Adrion[10] believes that glandular proliferation may occur here, but that owing to the loose stroma it does not give rise to the encapsulated tumour seen in other parts. In this series of autopsy specimens the changes of benign hypertrophy were present in the posterior lobe in 6 per cent (*see Figs.* 206, 207).

The histological evidence suggests that atrophy in the posterior lobe apparently occurs earlier than in the rest of the prostate. In the sixth decade, when the changes of benign hypertrophy may already be present in the lateral or middle lobes, the posterior lobe is frequently partly atrophic. This early atrophy may be *some explanation of the infrequency with which this lobe is involved by glandular proliferation.*

FIG. 198.—Atrophic acinus in posterior lobe showing loss of lining epithelium, peri-acinar lymphocytosis, and foreign-body giant cells applied to corpus amylaceum. Age 53.
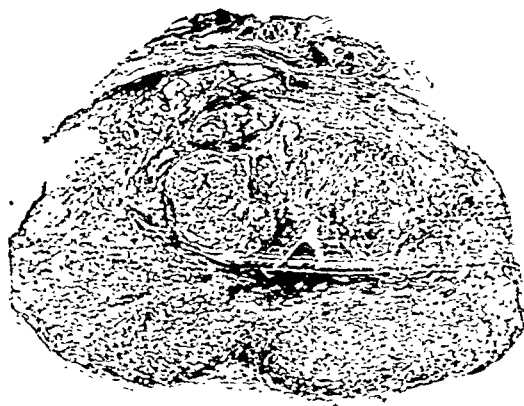
FIG. 199.—Nodules in both lateral lobes, some cystic. The lateral parts of the posterior lobe also show cystic changes, but posteriorly the acini are closely packed. Age 79.

The frequency of benign hypertrophy (88 per cent in men over 70 at autopsy) and its histology suggest an involutionary change common to the majority of men—a hyperplasia of some of the prostatic glands rather than a true neoplasm, which is followed in extreme old age by cyst formation.

2. The Fibrous Prostate.—Fibrous areas containing a varying amount of muscular tissue are frequently seen in the prostatic 'adenoma', and in a few cases constitute the bulk of the tumour. Their presence has led a number of authors to classify the tumour as a fibro-adenoma, or fibro-myo-adenoma.
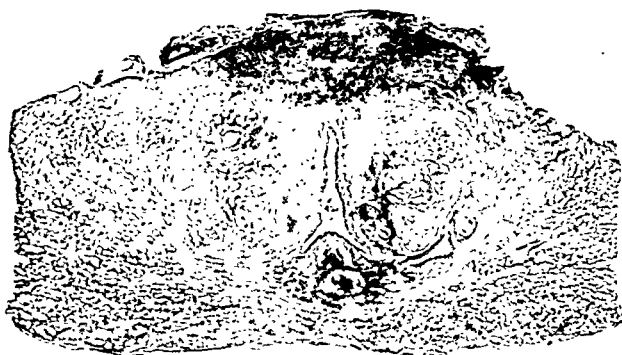
FIG. 200.—Section above utriculus masculinus. Nodules in lateral lobes. Age 68.

and they have been attributed either to a true neoplastic growth of the stroma or to an inflammatory change. The first view was that of Horn and Orator,[11] who believed that true myomata also occurred. Large areas of muscular tissue are not infrequently found in an 'adenoma', but in the normal prostate

FIG. 201.—Edge of nodule from *Fig.* 200, showing replacement of peripheral acini by connective tissue. The outline of the original nodule can still be seen.

many bands of plain muscle are present around the urethra, the amount varying in individuals, and at different levels in the same individual. Young,[12] Adrion,[13] and others believe that the connective-tissue overgrowth is the result of infection, and that epithelial elements are frequently present in these

fibrous areas. Walker[11] states that the fibrous prostate represents an essentially different pathological condition from glandular hypertrophy.

A number of prostates removed at autopsy and by prostatectomy showed these fibrous nodules, and in many cases stages in their formation could be

Fig. 202.—Further stage in the process of fibrosis. Age 61.

seen. A cellular fibrosis occurs around a glandular nodule, involving first the peripheral acini, which become compressed and finally obliterated. A fibrous nodule is left containing a number of arteriosclerotic vessels and

Fig. 203.—Complete replacement of glandular nodule by fibrous tissue. A number of arteriosclerotic vessels are present and the original capsule can still be seen. Age 65.

degenerate muscle fibres. The outline of the original glandular area still remains, though it no longer has such an encapsulated appearance (*Figs.* 200–203). The majority of these areas are commonly found in close proximity to the urethra, some covered only by mucosa, and may involve its whole length.

Overgrowth of connective tissue also gives rise to intra-acinar and intra-ductal papillomata with a thick connective-tissue stalk unlike the fine papillary processes usual in benign hypertrophy. They bear a close resemblance to the 'multiradicular' papillomata described by Cheatle[15] in the

FIG. 204.—Commencing fusion of papillomatous processes in fibrosis.

breast, occur most frequently in the neighbourhood of the urethra, and by the fusion of opposing papillomata may cause complete obliteration of the acinus (*Figs.* 204, 205).

FIG. 205.—Later stage of fusion from the same case as *Fig.* 204, showing more complete obliteration of acini.

Where fibrosis is marked, all the glandular tissue in proximity to the urethra is replaced by fibrous nodules. The muscular tissues in which the glands lie are also affected, and the urethra is 'walled' by fibrous tissue containing traces of glands and degenerate muscle. The involvement of Albarran's

glands may cause stenosis of the internal meatus, or a fibrous 'bar' at the bladder neck (*Figs.* 206, 208). In the presence of such fibrosis it is apparent that any glandular mass present will be exceedingly difficult to enucleate.
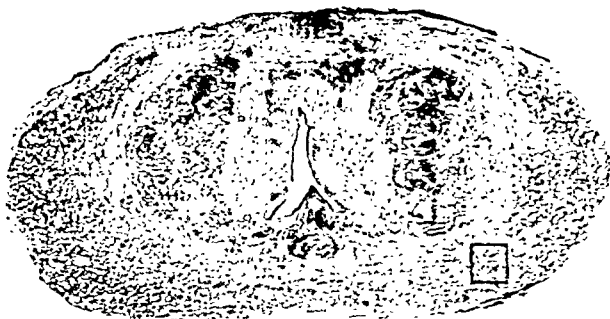
Fig. 206.—Glandular nodules in lateral lobes replaced near the urethra by fibrous tissue, in which the outline of some nodules is still visible. A small glandular nodule is also present in the posterior lobe. Age 68.

Thomson-Walker,[16] discussing the clinical conception of the 'fibrous' prostate, included in this term those prostates of 20 grm. or under. His figures give 3 per cent of prostates as showing hyperplasia of the stroma. In 5 per cent of the prostatectomies at the Middlesex Hospital the 'prostate'

Fig. 207.—The nodule in the posterior lobe outlined in *Fig.* 206. Atrophy of the surrounding acini is shown.

removed consisted mainly of fibrous or fibromyomatous tissue. Fibrosis was present though not predominant in 29 per cent. In those prostates removed at autopsy this fibrosis was present in :—

AGE		PER CENT
50–59	.. ..	10
60–69	.. ..	30
70 onwards	..	40

In two cases (2 per cent of prostates removed from men over 50) the change was sufficiently marked to classify them as 'fibrous' prostates, in both of which vesical trabeculation was present though there was no history of urinary symptoms.

The relation of a previous history of gonorrhœa is shown below.

Prostates with history of gonorrhœa, fibrosis in 47 per cent.
Prostates without history of gonorrhœa, fibrosis in 26 per cent.

Of 6 cases of prostatectomy in which the bulk of the prostate removed was fibrous, 2 had a history of gonorrhœa, one with stricture, and a third had chronic cystitis of many years' duration. These figures show that fibrosis

Fig. 208.—Fibrous nodule in Albarran's glands. From the bladder neck in the same case as *Fig*. 206.

is more common in, though by no means limited to, those cases in which a history of gonorrhœa is present. The absence of a history of gonorrhœa does not, however, exclude infection of some kind, nor does its presence necessarily indicate a previous prostatitis.

Acute inflammatory changes are present in the majority of prostates removed, particularly in that part of the 'adenoma' in close proximity to the urethra, and may be justifiably attributed to previous instrumentation. In 'fibrous' prostates chronic inflammatory changes are commonly marked. the perivascular lymphatics contain lymphocytes (*Fig*. 209), and the surrounding musculature is fibrotic. The other factor which bears an apparent relation

to fibrosis is age; whilst it is found in 10 per cent of prostates in the 6th decade, it is present in 40 per cent of those in the 8th.

The bulk of the evidence suggests that in a fibrous prostate we are not dealing with a separate pathological entity, but with a gland in which the
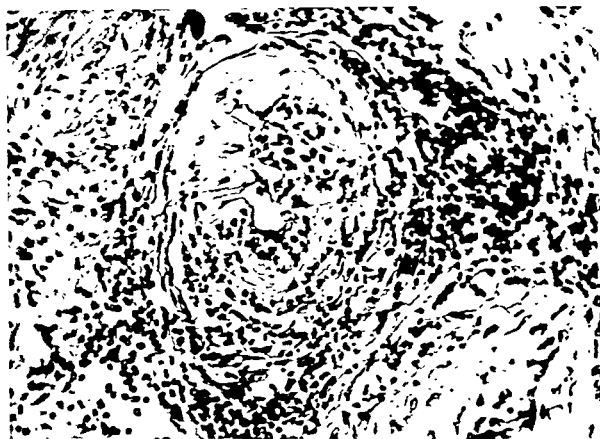
FIG. 209.—From a fibrous prostate, showing perivascular lymphocytosis. Age 61.

typical changes of benign hypertrophy are modified by fibrosis, due to chronic inflammation or to a degenerative change.

3. **Calculous Prostatitis.**—Small, gritty particles rarely larger than a pin's head are found in 40 per cent of prostates over 50. They are due to the deposit of mineral salts around corpora amylacea and in only a small number
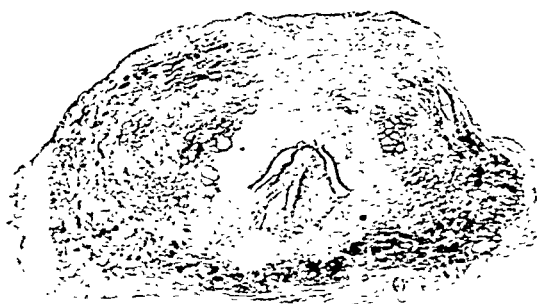
FIG. 210.—Section of calculous prostatitis. The atrophic appearance and the absence of prominent muscle bands is shown. Age 50.

are associated with inflammatory conditions. When inflammation is present the calculi are usually of larger size. Larger calculi, from 2 to 4 mm., occur in two conditions. In benign hypertrophy the ducts of the posterior lobe may contain calculi up to 1 to 4 mm. in diameter. These ducts as they pass

to the urethra occupy a horseshoe-shaped area behind the lateral lobes, and when these are enlarged in benign hypertrophy the calculi, if present, form a rim along the posterior aspect of the adenoma. Pressure on the ducts by the enlarging lateral lobes is a possible explanation of their presence.

FIG. 211.—Same case as *Fig. 210*. Some acini are atrophic while others show epithelial hyperplasia. Many of the ducts and acini contained pus cells.

The term 'calculous prostatitis' is, however, restricted to a definite type of prostate which was present in 5 per cent of autopsy specimens. Macroscopically the prostate is smaller than normal. In the specimen illustrated.

FIG. 212.—Calculi in a duct in calculous prostatitis. Age 58.

from a man aged 50, the size was equivalent to that of a boy of 15 (*Figs. 210. 211*). On section dark particles of varying size are scattered throughout the gland, with larger calculi in the neighbourhood of the urethra (*Fig. 212*). Microscopically the ducts and acini contain pus, and periductal lymphocytosis

and fibrosis are marked. The latter extends to the greater part of the stroma and musculature.

Gland destruction may be marked; the acini lose their high columnar epithelium and may show areas of proliferating darkly staining cells (*Fig.* 211). Krompecher[17] believed that these represented an inflammatory hyperplasia of the supporting cells of the epithelium and that the rare squamous carcinoma of the prostate arose in such areas. The histological picture is that of an atrophic gland, the result of chronic inflammatory changes, associated with calculi which are probably secondary to the infection (prostatitis, urethritis, stricture). Unlike benign hypertrophy, these changes are not localized to any one site but are present throughout the *whole* gland.

Of 14 specimens examined (autopsy and biopsy) benign hypertrophy was present in only 1, and here in only a small area. The relative absence of such changes can probably be attributed to the destruction of gland tissue which occurs.

CLASSIFICATION.

The material removed by suprapubic prostatectomy in the 114 cases under review has been classified into four groups, on the lines of the preceding description.

Benign hypertrophy
- *A.* *Glandular*, where the material removed consists of glandular tissue in its normal stroma.
- *B.* *Intermediate*, or mixed type where traces of fibrous overgrowth are present with glandular tissue.
- *C.* *Fibrous*, composed almost entirely of fibrous tissue.
- *D.* *Calculous prostatitis*.

The frequency of the four types was as follows :—

	PER CENT
Glandular	57·5
Intermediate	28·5
Fibrous	6·0
Calculous prostatitis	8·0

CLINICAL INVESTIGATION.

For the clinical part of the investigation as many of the patients who were able to come have been interviewed and examined ; others not able to come have filled in answers to a questionnaire. For those with whom contact could not be made recourse was had to the case notes alone. In this way the record of symptoms is based on 102 cases, that of mortality and post-operative inflammatory complications whilst in hospital on all the 114 cases, and the assessment of clinical results is made in respect of 68 cases who were either examined personally or who replied to the questionnaire.

Symptomatology.—The symptoms complained of by the patient, and their duration before seeking medical advice, are known in 102 cases comprising all four groups. In order of frequency they were as follows :—

	PER CENT
Increased frequency of micturition	82
Difficulty in micturition	58
Pain on micturition	37
Acute retention of urine	34
Hæmaturia	11

Other symptoms, such as incontinence, alterations in stream, and dribbling at the end of the act amounted to less than 10 per cent of the whole, and have not been included.

The average duration of symptoms before seeking advice in all cases was 3 years 2 months. In a number of cases, however, the first symptom of which the patient complained was an attack of retention of urine, so that its duration was only a matter of a few hours, whilst the longest period in which symptoms were present was 14 years; thus a figure representing an average duration for the series is of little value. The average age of the patients on seeking advice was 62 years 6 months.

When the types are considered separately the frequency of the different symptoms is as follows :—

SYMPTOM	TYPE OF PROSTATE			
	Glandular	Intermediate	Fibrous	Calculous
	Per cent	Per cent	Per cent	Per cent
Increased frequency	67	74	100	87
Difficulty	60	55	53	62
Pain ..	29	23	33	62
Acute retention	48	41	33	12
Hæmaturia ..	20	13	0	12
	Years	Years	Years	Years
Average duration of symptoms	2	$2\frac{3}{4}$	$5\frac{1}{3}$	$2\frac{3}{4}$
Average age	63	65	64	58

Increased frequency of micturition was a constant feature in the fibrous type, in which type also there was a longer average duration of symptoms: this type, indeed, produces a picture somewhat resembling that of urethral stricture, the symptoms being due not to a glandular tumour, but to a progressive fibrosis surrounding the urethra and involving the muscles.

Difficulty in the act was present to about the same extent in each type, but pain was a more prominent feature in the calculous group. Acute retention was most common in the glandular and intermediate types, and in more than half of the cases of these groups in which it occurred there was more than one attack, relief by catheterization being followed by intervening periods of comparatively easy micturition. In the fibrous and calculous types, on the other hand, when acute retention did occur one attack was the rule, operation being necessary for its relief. In accordance with expectations hæmaturia was commonest in the glandular type, and was absent altogether in the fibrous prostates.

Post-operative Inflammatory Complications. — Under this heading are included, in order of frequency, epididymitis, secondary hæmorrhage, and ascending renal infection. The total of such complications was 34, an incidence of 29 per cent for the series. When the different histological types are considered separately the incidence of complications is seen to be relatively low in the glandular type, but considerable in the other three, with a gradual increase from the intermediate group to the cases of calculous prostatitis.

TYPE	TOTAL	COMPLICATIONS
		Per cent
Glandular	66	16
Intermediate ..	33	42
Fibrous	6	50
Calculous	9	66

Considered in more detail the inflammatory complications were as follows :—

Glandular.—
Epididymitis	7
Secondary hæmorrhage ..	2
Ascending infection	2
	—
Total ..	11 out of 66 (16 per cent)

Intermediate.—
Epididymitis	9
Secondary hæmorrhage ..	4
Ascending infection	1
	—
Total ..	14 out of 33 (42 per cent)

Fibrous.—
Epididymitis	3
Secondary hæmorrhage ..	0
Ascending infection	0
	—
Total ..	3 out of 6 (50 per cent)

Calculous Prostatitis.—
Epididymitis	4
Secondary hæmorrhage ..	(1) occurring in one of the cases with epididymitis
Ascending infection	2
	—
Total ..	6 out of 9 (66 per cent)

Whilst the total number of cases is small, particularly in the fibrous and calculous types, the difference in incidence of complications is striking.

Epididymitis occurred either on one or both sides in 23 cases, or 20 per cent of the total. This figure approximates to that given by Randall[18] of 23 per cent, but is considerably less than that of Winsbury-White[19] (82 per cent). which, however, included the very mildest forms. It is sufficiently high to make division and ligature of the vasa a desirable addition to the operation, as advised by Morson.[20] That ligature alone is not sufficient was shown in one case in this series where epididymitis followed on the side ligatured but not on the side divided. The time of onset varied from a few days after operation to some weeks after leaving hospital; one case went on to abscess formation, and the remainder subsided under expectant treatment.

Secondary hæmorrhage occurred in 7 cases (6 per cent). It usually started about ten days after operation, and was responsible for two deaths. Ascending infection leading to pyelonephritis was the least common but most fatal complication : it occurred in 5 cases (4·4 per cent) and caused death in each instance. The possibility of the occurrence of minor degrees of ascending infection which did not prove fatal cannot be excluded.

The relationship between the histological type and the incidence of these complications is probably indirect, depending in part on the greater trauma inflicted in attempting to remove a fibrous or calculous prostate than one which is purely glandular, and in part on the lighting up of a previous chronic infection in the prostate.

Mortality.—The number of deaths from the operation was 13 (12 per cent), which compares favourably with the figure of 19·5 per cent for twelve general hospitals quoted by Thomson-Walker.[21] Figures are given for each histological type, but the numbers are too small to warrant any general conclusions.

TYPE	MORTALITY
Glandular	7 out of 66 (10 per cent)
Intermediate	4 out of 33 (11 per cent)
Fibrous	0 out of 6 (0 per cent)
Calculous prostatitis	2 out of 9 (22 per cent)

The cause of death was as follows :—

Glandular (7 cases)	Pyelonephritis, uræmia	2
	Primary hæmorrhage	2
	Secondary hæmorrhage	1
	Pulmonary embolus	1
	Myocardial degeneration	1
Intermediate (4 cases)	Pyelonephritis, uræmia	1
	Primary hæmorrhage	1
	Secondary hæmorrhage	1
	Syncope	1
Fibrous		0
Calculous prostatitis (2 cases)	Pyelonephritis, uræmia	2

Fatal primary hæmorrhage thus only occurred after prostates with glandular elements had been removed, but not in the fibrous and calculous varieties. It is recognized that death is as a rule due to a combination of causes in these cases, and the discrepancies in different mortality tables have already been pointed out by Walker.[22]

Clinical Results.—The assessment of the results of the operation has been arrived at by a consideration of symptoms and by a complete examination of the patient whenever possible. From the symptomatic point of view the operation has been considered completely successful only if the patient was living in entire comfort as regards his urinary function, soundly healed, with complete control, able to pass a good stream without pain or difficulty, and not having to micturate more than twice during the night. Such cases are classified as 'normal', and on this basis the percentage of normal results in each of the four histological groups is found to be as follows :—

TYPE	NORMAL RESULTS AFTER OPERATION
Glandular	29 out of 33 (88 per cent)
Intermediate	16 out of 22 (73 per cent)
Fibrous	1 out of 6 (17 per cent)
Calculous prostatitis	3 out of 7 (43 per cent)

Allowance must be made for the necessarily small number of cases of fibrous prostate and calculous prostatitis, but in this series it appears that a normal result after operation is five times as common in the glandular type as in the fibrous type.

The results in each group will now be considered in more detail.

Glandular Type (33 cases reviewed)—
Normal result   in 29 (88 per cent)
Increased frequency   in 3
Nocturnal incontinence   in 1

In general these patients were very well, and extremely pleased with the result of the operation. In three-quarters of the cases examined post-operatively the prostate was appreciable and showed adenomata on cystoscopy and rectal examination, and mild cystitis with some trabeculation was present in half the cases, although in the majority the urine contained no pus and was sterile on culture. In one the prostatic enlargement was considerable, although the patient had no symptoms. One patient had a return of difficulty in starting and frequency of micturition a year after operation, and it was found at this time that there was stenosis of the internal urinary meatus, so that only a filiform bougie could be passed. Gradual dilatation restored his function to normal, and cystoscopy showed irregularity of the prostatic margin. Of the three patients with increased frequency of micturition none had to pass water more than thrice in the night ; one of these was a man of 76 who had had a large diverticulum of the bladder containing eighty-nine stones ; this was excised seven weeks before prostatectomy was undertaken. One patient, a man of 75, found he could not hold his water by night, and therefore wore a collecting apparatus. The highest amount of residual urine was 3 oz.

Intermediate Type (22 cases reviewed)—
Normal result   in 16 (73 per cent)
Increased frequency   in 5
Permanent suprapubic fistula in 1

In general the results were again good in this series. Prostatic adenomata were present on rectal and cystoscopic examination in half the cases examined, and some degree of cystitis in more than half, with some pus cells and *B. coli* in the urine. In one case the prostatic enlargement was considerable. The degree of increased frequency of micturition in the cases enumerated was higher than in the purely glandular type. There was stenosis at the internal meatus in two cases ; in one of these the passage of the cystoscope caused the rupture of a band at the meatus, and his frequency of micturition was subsequently reduced from nine times at night to once, starting the act being also made much more easy. In the other, a frail man of 76, in whom out-patient instrumentation was not considered desirable, there was sufficient stenosis to produce a permanent suprapubic fistula for which an apparatus was worn. The highest amount of residual urine was $3\frac{1}{2}$ oz.

Fibrous Type (6 cases reviewed)—
Normal result   in 1 (17 per cent)
Increased frequency   in 2
Delayed healing   in 2
Sensation of incomplete emptying in 1

The final result in these cases was more satisfactory than the figures suggest, but only one could be classed as normal, and in this case there was

considerable scarring and injection around the internal meatus. In general the degree of cystitis present was more severe than in the previous groups. and in one case there was marked cystitis cystica around the internal meatus, which probably accounted for his sensation of incomplete emptying of the bladder. There had been sufficient stenosis of the internal meatus to delay healing for more than six months in two cases, and in a third the regular passage of bougies had been necessary for some weeks after operation. On rectal examination the prostate in all these cases was either flat or concave. and there was no evidence of new adenomatous formation. The highest residual urine was 1 oz.

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Calculous Prostatitis (7 cases reviewed)—

Normal result	in 3 (43 per cent)
Increased frequency	in 2
Nocturnal incontinence	in 1
Death elsewhere two years after operation*	in 1

*Inquiry elicited the information that this patient had obstruction at the internal meatus.

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The final results in this group were numerically slightly better than in the purely fibrous type, but residual cystitis was rather severe in some cases. One patient who was getting up four times at night to micturate had a persistent epididymitis five months after operation and his condition at that time was rather worse than before operation ; one had residual calculi. The patient with nocturnal incontinence had a very severe cystitis, the infecting organism being *B. pyocyaneus* ; this organism was present before operation in the bladder urine. Stenosis of the internal meatus of some degree was present in two cases. and the presence of adenomata was noted in only one. and these were small and in the anterior lobe. By rectal examination the prostates were appreciable as flat or apparently normal prostates and in no case felt enlarged. The highest residual urine was $2\frac{1}{2}$ oz.

DISCUSSION.

Examination of these cases showed that the final result was very good in those cases with purely glandular prostates, and good in the intermediate type, whilst in the fibrous and calculous cases it was much less satisfactory. The factors causing the poor results in the two latter groups were an increased liability to stenosis of the internal meatus and a more frequent and severe residual cystitis ; these may both be considered inevitable results of the difficulty in removing all the fibrous tissue which commonly extends throughout the prostatic urethra in the fibrous type, and the impossibility of taking away all the infected gland in calculous prostatitis.

Re-appearance of the Prostate.—On the other hand, the re-appearance of the prostate was more marked in the glandular and intermediate types. and in two of these there was sufficient enlargement to suggest that further obstructive symptoms might ensue and necessitate a second prostatectomy. In the fibrous and calculous types post-operative rectal examination showed a flat or concave surface to the examining finger, and cystoscopy showed no adenomatous formation except in the one case already mentioned with small anterior adenomata.

Davies and Loughnane[23] found that of 19 prostatectomized patients. examined with the urethroscope only 2 still showed total absence of the prostate. In one of these it is recorded that there was no prostatic enlargement felt per rectum before operation, from which it appears a possible conclusion that the case was one of fibrous prostate ; in the other case no note is given of a pre-operative rectal examination.

Amount of Prostate removed at Operation.—It has already been shown that the posterior lobe is but rarely involved in the glandular changes of benign hypertrophy, but is subject to early atrophy and compression in lateral-lobe enlargement, and that it is partially separated from the remaining lobes by a layer of plain circular muscle continuous above with the vesical sphincter. This supports the view first advanced by Wallace[24] that suprapubic prostatectomy in the glandular or early intermediate types really consists of enucleation of the lateral, middle, and anterior lobes only, i.e., the peri-urethral glands as previously defined. The posterior lobe is probably only removed, and then only partially, when there is sufficient fibrosis to destroy the natural plane of separation between it and the remaining lobes, as may occur in the purely fibrous and calculous types.

Other Methods of Treatment.—In the glandular and intermediate types of benign hypertrophy the results in this series are in accord with the general conclusion of Walker that some 90 per cent of patients will survive the operation of suprapubic prostatectomy ; complete urinary relief may be expected in from 70 to 90 per cent of cases. There remain the cases of fibrous prostate and calculous prostatitis in which some other procedure might be considered. The possible alternatives to suprapubic prostatectomy appear to be :—

1. *Catheter Life.*—This has many well-recognized disadvantages and should be necessary only in a small and decreasing number of cases.

2. *Regular Dilatation.*—This is of service in a limited number of cases, particularly of fibrous prostate.

3. *Perineal Prostatectomy.*—This has never been widely practised in England, and even in America its popularity seems to be on the wane.

4. *Per-urethral Partial Prostatectomy.*—If the results already claimed for it are borne out by those of larger series of cases[25] this appears to be the most hopeful procedure, certainly for the fibrous prostate and possibly for calculous prostatitis.

Recognition of Cases.—The recognition of the type of prostate encountered is thus of absolute importance ; fibrous and calculous cases present a clinical picture with many common features, and in the latter decisive confirmatory evidence may be obtained by radiological examination or, as sometimes happens, by the striking of a loose calculus on instrumentation. The main diagnostic features of the fibrous prostate are discovered from the history. symptoms, and physical and endoscopic examinations, and the outstanding points as compared with glandular prostates, are here summarized :—

History.—Relatively long duration of symptoms ; possible previous infection. e.g., gonococcal.

Symptoms.—Increased frequency of micturition the rule, hæmaturia the exception. Previous attacks of acute retention uncommon.

Rectal Examination.—Little enlargement of the prostate; induration without infiltration. Examination with a bougie in the urethra helps one to distinguish the induration of a fibrous prostate from the elasticity of a glandular enlargement on the one hand and the stony hardness of a carcinoma on the other.

Cystoscopy and Urethroscopy.—Some resistance to the introduction of the instrument in the prostatic urethra; little intravesical projection of the prostate, but possibly a small projecting collar, raised more posteriorly than anteriorly, or a posterior bar; cystitis and trabeculation; prostatic urethra may be shortened, and does not show the projecting lateral lobes of typical glandular enlargement.

Urine.—May be infected or contain threads. Moderate residual. A distinction between a fibrous prostate and calculous prostatitis may only be possible after X-ray examination. but pain on micturition is a more prominent feature of the latter.

SUMMARY.

1. An investigation has been undertaken to correlate the structural appearances and clinical features in non-malignant prostatic obstruction.

2. The appearances of 138 autopsy prostates have been studied, and the following histological classification is suggested : (*a*) Glandular enlargement. (*b*) Intermediate form, with some fibrosis in the glandular tissue. (*c*) Fibrous prostates. These three are all examples of benign hypertrophy. (*d*) Calculous prostatitis, in which calculi are associated with chronic inflammatory changes in an atrophic gland.

3. The prostates from 114 consecutive suprapubic prostatectomies (non-malignant) have been examined and classified.

4. The clinical features of these cases have been reviewed and certain differences in the four types noted. The symptoms differ somewhat in each type. Post-operative inflammatory complications are less common in the glandular than the fibrous prostates, and greatest in calculous prostatitis. The mortality of the operation is highest in calculous prostatitis. The end-results are excellent in the glandular type, but less satisfactory in the fibrous and calculous types.

5. Re-appearance of the prostate after operation is the rule in the glandular types, but not in the fibrous.

6. Per-urethral partial prostatectomy is probably the operation of choice for the fibrous prostate, and possibly for calculous prostatitis.

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A RUBBER ŒSOPHAGUS.

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TWENTY-THREE years ago, for an extensive carcinoma, I removed the larynx, part of the pharynx, the cervical œsophagus, and as much of the thoracic œsophagus as my fingers could reach behind the sternum. This, of course, necessitated a permanent tracheotomy and a permanent gastrostomy. The patient is alive and well to-day.* She takes her food by mouth, masticates and swallows it, and no on-looker would guess there was anything unusual in the process—but an extrathoracic rubber œsophagus has replaced the normal one (*Figs.* 213, 214.)
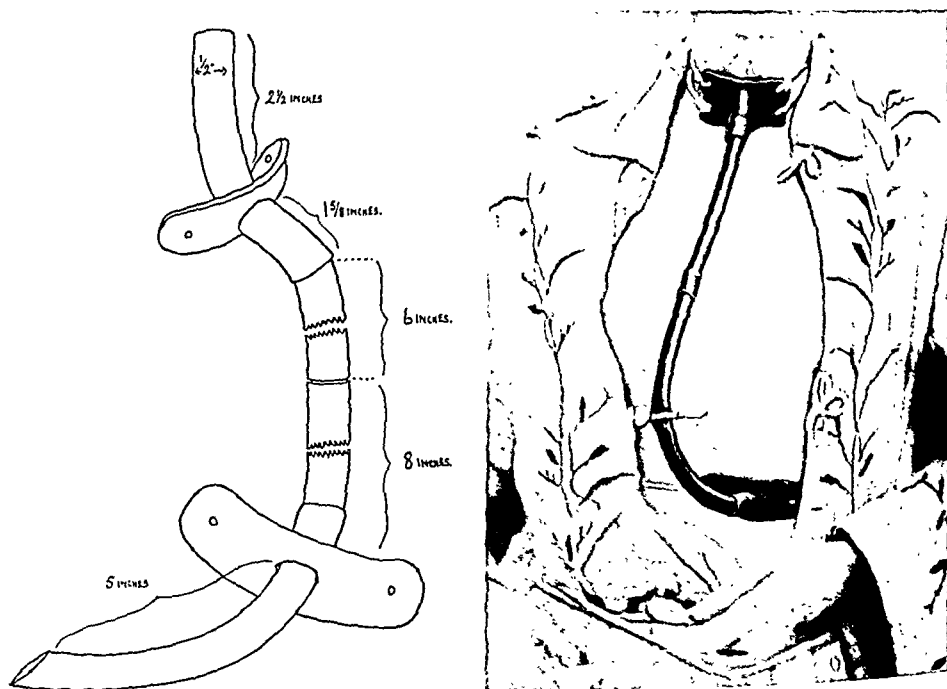
FIGS. 213, 214.—Tracheotomy tube and rubber œsophagus as worn by the patient.

The patient, a female aged 40, was first seen on Sept. 19, 1909. She complained of hoarseness and increasing difficulty in swallowing solids: this

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* This patient was shown and the operation described at the Clinical Section of the Royal Society of Medicine on Nov. 12, 1909; and the patient shown wearing the rubber œsophagus at the Seventeenth International Congress of Medicine, London, 1913.

had been noticed for about five months. Laryngoscopic examination revealed a large malignant mass filling up the lower part of the pharynx, and extending so far forwards that it covered over a large portion of the vocal cords, and almost completely hid the left; one could, however, get a glimpse of the left cord, and it was fixed; the right vocal cord moved freely. By laryngoscopic examination it was not possible to state whether the mass of growth was only postcricoid or whether it extended round the whole circumference of the upper part of the œsophagus. There was no obvious glandular enlargement in the neck. The patient was markedly emaciated.

The malignant nature of the condition was explained to the patient's friends, and also the fact that the complete removal of the growth was considered practically impossible. It was suggested that she should be allowed to remain unoperated upon. and that when the disease had so far advanced that any trouble was experienced in swallowing fluids, gastrostomy should be performed. The risks of an attempt at extirpation were pointed out, and the unenviable condition of the patient, after even a successful operation. was described.

The patient returned on Sept. 25. 1909, and at the urgent request of the patient and friends I consented to attempt the removal of the growth.

Sept. 28, 1909.—Gastrostomy was performed, Abbe's modification of Kader's method being adopted.

Oct. 2, 1909.—An incision was made extending from the hyoid bone to within one inch of the suprasternal notch. The isthmus of the thyroid gland was divided in the middle line. I was anxious to make a close examination of the extent of the growth, and hoped to find I could save some part of the larynx, and maybe, a strip of œsophagus; so I inserted a tracheotomy tube. and then slit up the thyroid cartilage. It was now found that the malignant growth had invaded the whole of the upper orifice of the œsophagus, and a good deal of the pharyngeal wall, and to remove the disease it was necessary to remove the larynx and the upper part of the œsophagus.

The trachea was cut across obliquely, so as to save as much as possible of the posterior wall, a transverse skin incision was made at the suprasternal notch, and the portion of skin between this and the longitudinal incision undermined—through this the trachea was brought and stitched to the skin.

On cutting through the hyoid bone and making a transverse incision immediately below it. a good exposure was obtained of the pharyngeal growth. and it was easy to incise the pharyngeal wall above the growth, and with very little difficulty the lateral walls of the pharynx were freed, and the pharynx with the larynx were separated from above downwards; at the level of the cut trachea it was obvious to the fingers manipulating the œsophagus that the malignant disease extended lower down, so the œsophagus was isolated still lower. and a strong silk ligature passed round it at a level which felt well below the disease; the œsophagus was cut through, and the growth removed (*Figs.* 215, 216).

On slitting up the œsophagus it was evident that the section had been made too near the growth. so the stump was drawn upon by the encircling ligature, the ends of which had been purposely left long, and the œsophagus

was separated for another inch—which was as far as the fingers could reach within the thorax—here the œsophagus was ligatured and cut across.

The transverse incision, and all of the median—save the lower inch—were now closed with one layer of sutures and the interior packed lightly with gauze. The convalescence was quite uneventful.
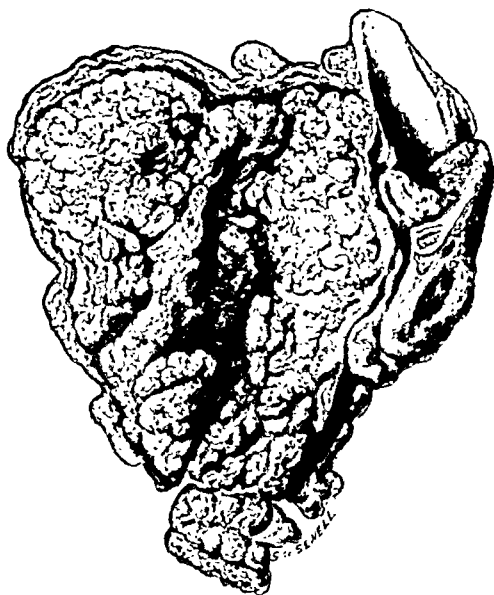
FIG. 215.—The growth viewed from behind when the pharynx and œsophagus had been opened in the mid-line posteriorly.

Oct. 14, 1909.—The secretions from the mouth, which trickled out of the unclosed part of the wound, came on to the surface immediately above the tracheal opening, and had constantly to be prevented from being sucked into the trachea. I therefore sewed up the lower inch of the opening, and extended it upwards for a corresponding distance.

I had another reason for wishing to alter the position of the pharyngeal sinus to one higher up in the neck, in that I had designed a small celluloid cup, edged with a rubber cushion, which I attempted to keep in close apposition to the opening by means of a rubber band which encircled the neck, and, this cup being connected by a tube with the gastrostomy opening, I hoped it would serve as a means of conducting food and the secretions of the mouth to the stomach.

This arrangement, however, did not work well; in order to prevent leakage it was necessary to fasten the elastic band so tightly round the neck that the patient suffered great discomfort, and even in this new position the

FIG. 216.—Section of growth showing a squamous-celled carcinoma.

pharyngeal opening was too near the tracheal opening, so that any leakage was at once sucked into the trachea, and set up a distressing cough.

Nov. 1, 1909.—I closed the sinus in the lower part of the neck, and made a new one immediately below the hyoid bone ; to make this opening permanent, I fashioned four flaps from the surrounding skin, and with them lined the walls of the sinus. Through this opening I passed into the upper pharynx the short end of a rubber empyema tube, using the largest that could be introduced, and this was kept in position by means of a piece of tape passed round the neck and tied into the holes in the flanges of the empyema tube ; the long end of the empyema tube was connected with the gastrostomy tube. At the time of this operation I was not able to detect any enlarged glands in the neck.

What remains to be told had best be done by quoting from letters received.

Nov. 24, 1909.—" The swallowing tube does not act well, I am sorry to say, but I hope it will become better as the wound heals."

Dec. 1, 1909.—The sister writes : " All last week the swallowing tube could not act, and we were bitterly disappointed, but since Sunday the scene is changed. Yesterday she had minced pheasant and bread sauce, and vegetables for dinner, then stewed pears and cream, all of which she thoroughly enjoyed. Bread and butter she has, milk, chocolate, and in fact, whatever happens to be going, all by mouth ! "

April, 1910.—" The week after Easter I was able to get weighed, and I had gained over a stone since I was in London ".

July, 1910.—This patient sent me her wedding card !

Feb. 23, 1911.—" I am glad to tell you I am feeling very fit indeed. If it were not for my cough, which still bothers me, I could say I never felt so well in my life. My whisper seems to be stronger ; I do not experience any difficulty in doing my shopping. The swallowing tube acts very well. Some days it is really perfect. When I tell you I went to dinner at the —— Hotel the last time I was in B—— with my husband, and dined in a room with dozens of people, you will agree with me it is very wonderful. Of course, it is not always so well behaved, but I have very little to complain of. I have my cousin staying with me now, and every day we have had long walks of three and four miles—this will tell you I am strong."

March 8, 1912.—" I am sending you a set of tubes to see. The tape passes round my waist, and this keeps the gastrostomy tube in better. I only take this tube out three times a week to boil ; it is always quite pure and clean ; there never has been any unpleasant smell, or anything disagreeable in connection with it. The throat tube I boil every morning with the tracheotomy tube. I take the latter out at night as well, but only clean it. I find once a day is quite sufficient to boil that. I always wear lace round the throat to breathe through. I have all my dresses cut low in front to suit the tracheotomy tube, and fill in with lace." (*Fig.* 217.)

Oct. 3, 1912.—" I am sending you a snapshot of myself—I thought you would like to see how robust I look (*Fig.* 217). I am feeling so well, and am getting quite fat. I don't know whether you will laugh when I tell you

that I really think there must be some development taking place in the throat, because I discovered last week that I was able to blow out a match—a thing which would have been utterly impossible twelve months ago."

FIG. 217.—Photograph of the patient, showing the style of dress adopted.

April 16, 1913.—" I am feeling very fit indeed. My cough is troublesome sometimes, especially if I take cold, but with that exception I have very little to complain of. My quiet country life suits me, and my people all think I am looking the picture of health. I only have to do my throat twice a day, and the swallowing tube acts very well. The gastrostomy incision is perfectly white and healthy looking. I only take out that tube once a week now, and then it is perfectly clean and there is never anything unpleasant. Everybody at home, thinks my whisper *much* stronger and clearer than when I was home last, and I think it must be, as I never have any difficulty in doing my shopping."

July 9, 1932.—" I am thankful to say I am very well. I have to be careful not to take cold, and if I do a bit too much the tube chafes the entrance to the stomach, and I have to rest a day; but I really lead quite a normal life. We have a large garden and last summer I used up all our fruit by making over 100 lb. of jam—so you see I am not an invalid.

" I often go into B—— for a day's shopping and lunch at a restaurant —so the swallowing tube acts well. No one seems to notice my whisper; and I get no difficulty in being heard, unless it is *very* noisy. Sometimes people say ' What a cold you have ', and suggest many remedies, which amuses me greatly.

" I do a bit of Church work too—I am treasurer for our Parish Magazine, a member of the Church Council, District Secretary for the Girl Guides, and do the Altar flowers. I have not had to call in my doctor for three years."

With regard to the rubber œsophagus now being used by the patient, the original empyema tube has been replaced by a curved rubber tube fitted with a flange—its shape is seen in the illustrations (*Figs.* 213, 214). The Jacques catheter originally introduced into the stomach at the time of the gastrostomy, and whose size was No. 9, has been at various times replaced by rubber tubes of larger and larger size, and eventually has attained its present size, a red rubber tubing bore No. 15; this too is fitted with a flange, 5 in. from its lower end, and to this tapes are attached; the tapes being tied around the abdomen keep the gastric tube in position.

OCCLUSION OF THE MAIN ARTERY AND MAIN VEIN OF A LIMB.*

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INTRODUCTION.

LIGATION of the main vein of a limb has acquired in recent years a notable popularity among some surgeons, who have employed it as a measure for the relief of certain disturbances of the circulation in the limb. The measure has been applied with particular zeal to conditions of deficient supply of arterial blood, and such application is even accorded the dignity of a surgical principle, usually spoken of as 'restoration of the balance of the circulation'. Furthermore, ligation of the main vein is believed to exert its benefits most fully when an immediate severe deprivation of arterial blood has followed a sudden occlusion of the main artery. Such circumstances, as is well known, are fraught with the gravest possible consequences to the function and life of the limb, and the choice of treatment is one of immediate and vital importance. The question of the application of venous ligation is obviously, therefore, not trivial. The present work is an experimental study of the effects of venous ligation in sudden arterial occlusion.

The origin of the practice of deliberate venous ligation in recent times has been so thoroughly traced by Brooks[5] as to make a detailed historical review unnecessary here. Ligation of the main vein of a limb probably owes its present standing as a therapeutic procedure very largely to the undoubted success which attended its use when the main artery had been ligated for the cure of an arteriovenous communication in the main vessels. The condition of arteriovenous communication is not considered in this paper. Venous ligation has also been employed in conditions of chronic arterial disease.[10, 11, 15, 16] Reports of the results are conflicting, and the benefits seem to be temporary and uncertain.

There is, however, a widespread and hitherto unquestioned belief that, if the main artery of a limb be suddenly occluded, as, for example, by necessary ligation, then ligation of the main accompanying vein lessens the chances of subsequent development of gangrene. Certain clinical and experimental observations have been cited to justify the belief. The most valuable and suggestive evidence on the clinical side is furnished by the personal experience of Sir George Makins[12] in the treatment of wounds of large vessels

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during the war. The main experimental support is the work of Brooks and Martin,[3] and an extension of their experiments by Holman and Edwards,[7] who claimed that ligation of the vein at a level higher than arterial ligation still further reduced the incidence of gangrene.

The favourable results of simultaneous venous ligation seem to have been universally accepted. The mechanism whereby gangrene is diminished is still, however, disputed. Many theories have been submitted in explanation, together with experimental results frequently conflicting and contradictory. A brief summary of these views and investigations is inserted at this point.

All workers are agreed that after occlusion of the main artery occlusion of the main vein causes a rise in pressure in the arteries distal to the obstruction. It was found that the pressure rose also in the veins and to a greater extent than in the arteries.[3] All recorded experiments on ligation of the vein at a level higher than the arterial occlusion (proximal venous ligation) show that the higher the level at which the vein is ligated, the greater is the rise in limb arterial pressure.[7, 17] The temperature of the tissues of the distal part of the limb falls after arterial occlusion, and the fall is accentuated by subsequent occlusion of the vein.[3]

Experiments which were quoted as indicating that the volume flow of blood per minute through the limb was increased by ligation of the vein were destructively criticized by Brooks. By direct methods the approximate total volume of blood flowing through the whole limb in unit time has been measured by Montgomery,[14] who found that proximal venous ligation caused a considerable reduction of volume flow. His experiments have been repeated and amplified in this investigation, and their significance is discussed later. Injection of the vessels at intervals after vascular occlusion shows that the calibre of the distal arteries is increased for some time by venous ligation.[17, 18, 19] There is some discrepancy in the estimates of the time during which the calibre remains increased.

Four main theories have been propounded to explain the beneficial effects of venous ligation :—

1. The theory that ligation of the vein 'restores the balance of the circulation'. This implies a belief that, in occlusion of the artery only, the capacious main vein affords a too ready outlet for the diminished quantity of blood reaching the part below the occlusion. The blood tends to supply mainly the proximal tissues of this part and is not dispersed to the distal tissues. There is thus a condition of imbalance of inflow and outflow, which, it is claimed, can be corrected by venous occlusion.

A closely allied idea is that venous ligation keeps the blood in longer contact with the tissues, which are, presumably, enabled thus to extract more oxygen from it.

2. The theory, formulated by Brooks, of a more homogeneous distribution of the arterial blood. The main favourable influence, according to him, is the raised intravascular pressure, which serves to keep the capillaries patent in areas in which they would otherwise collapse, and thus to distribute the arterial blood more uniformly throughout the tissues.

3. The theory of the development of a 'richer collateral bed' is the

consequence of injection experiments. The exact significance of the increased size of the vessels is not clearly stated.

4. The theory of a 'masked sympathectomy' is advanced by Leriche and Fontaine.[9] They believe that the initial rise of limb arterial pressure after venous ligation is associated with a transient peripheral vasoconstriction, which is succeeded in some minutes by an active vasodilatation, and that these effects are produced by reflex action along sympathetic nerve fibres.

Our experiments began with a study of the effects of temporary venous occlusion on the incidence of gangrene caused by arterial obstruction. Evidence of beneficial results from permanent or temporary occlusion of the main vein, however, was not forthcoming. A comprehensive re-investigation of the subject seemed desirable and will now be reported.

SURVIVAL EXPERIMENTS ON LIGATION OF ARTERIES AND VEINS.

DESCRIPTION OF EXPERIMENTS.

These experiments were carried out on rabbits. The total number for consideration is 109, divided into seven series :—

Series 1.—Ligation of the external iliac artery : 10 animals.

Series 2.—Ligation of the common iliac artery : 10 animals.

Series 3.—Ligation of the common and external iliac arteries (both arteries) : 30 animals.

Series 4.—Ligation of both arteries and of the common iliac vein : 30 animals.

Series 5.—Ligation of both arteries and of the inferior vena cava : 10 animals.

Series 6.—Ligation of both arteries and temporary ligation for twenty-four hours of the common iliac vein : 11 animals.

Series 7.—Ligation of both arteries and temporary ligation for twenty-four hours of the inferior vena cava : 8 animals.

Series 1 afforded a study of the effects of mild ischæmia. *Series* 2 and 5 were a test of the results of Holman and Edwards, while *Series* 3 and 4 formed a repetition of the work of Brooks and Martin but in a larger series of experiments. *Series* 6 and 7 were an investigation of temporary venous obstruction. A common assumption which holds that the beneficial effects of venous occlusion are exercised during the first twenty-four hours is thus subjected to experimental trial.

The vessels of the right posterior extremity were exposed by a mid-line abdominal incision. Arteries were divided between ligatures, veins were ligated. Temporary venous ligation was carried out in the following way. A piece of vaselined braided silk was passed through muscle on each side of and below the vein, and tied in a loop ligature. The vessel wall was thus protected by muscle from direct pressure of the ligature. The long end of the loop was brought out through a stab-wound in the flank and the ligature was released and removed by traction on the end at the required time. All cases in which constriction of the vein from fibrosis was found subsequently are omitted from consideration. The animals were kept alive for periods as long as 230 days.

EFFECT OF VASCULAR LIGATIONS ON FUNCTION AND STRUCTURE OF THE LIMB.

The animals have been divided into four groups as follows: *Group I*—Those which developed gangrene. *Group II*—Those with severe circulatory disturbances, but without gangrene. *Group III*—Those with moderate circulatory disturbances, without gangrene. *Group IV*—Those with slight and apparently temporary circulatory disturbances, without gangrene. Since the changes for any one group were almost identical in all series, each group is discussed without special reference to the vascular ligation. The description of the changes is necessarily brief.

Group I.—In this group there was death of the skin or of the skin and deeper structures together. Gangrene was of three main types: *Type A*—Massive gangrene, involving all the tissues of a portion of the limb, such as the whole foot or leg below the knee. *Type B*—Late gangrene of peripheral parts, such as the digits or portions of digits. *Type C*—Very late and limited gangrene of the peripheral parts. One case of gas gangrene occurred; all others were cases of dry gangrene.

Type A.—The features of dry gangrene became apparent between the second and seventh days. From the commencement the whole limb was powerless, cold, and of diminished volume, and the tendon reflexes were absent. Swelling appeared after a period of between sixteen and forty-eight hours and was limited to the portion ultimately surviving. It was more marked after venous ligation and especially after ligation of the inferior vena cava. The muscles which ultimately died *en masse* responded to direct stimuli on occasion for as long as eight hours after ligation. Sometimes they passed into a condition resembling rigor mortis between four and ten hours.

At post-mortem interpretation of gangrene was not always easy during the first forty-eight hours, and all cases of doubtful gangrene are included in *Group II*. In the surviving portion there were œdema, subcutaneous and intermuscular, congestion of superficial vessels, and, in the muscles, diffuse changes which became distinctive only after forty-eight hours. Most muscles had undergone necrosis which involved the whole or part of the muscle. The appearance was that of an anæmic infarct, the pale yellow necrotic area being separated from living muscle by a mottled reddish demarcation zone. Necrotic muscle fibres were swollen, disintegrated, and structureless, separated by fluid and cellular débris, and the nuclei did not stain. The demarcation zone was characterized by dilated small vessels and by a proliferation of leucocytes among degenerated fibres. Some of the fibres had a deep basic stain, after the third day, from deposition of calcium.

Type B.—In the early stage the changes during life were similar to those in *Type A*. Swelling was generalized but most marked in certain areas. Those were found later to contain necrotic muscle. Later, during the second week, as swelling subsided, such areas became indurated. Gradual return of voluntary power began during the second week, but was masked later by the development of *contractures*, which from the third week onwards steadily produced fixation at the joints, and finally converted the limb into a rigid

deformed appendage (*Fig.* 218). Gangrene never appeared before the fourth week. It affected the digits only, and was preceded by changes in the skin, such as loss of hair, discoloration, and ulceration. Gangrene, once developed, frequently spread proximally.
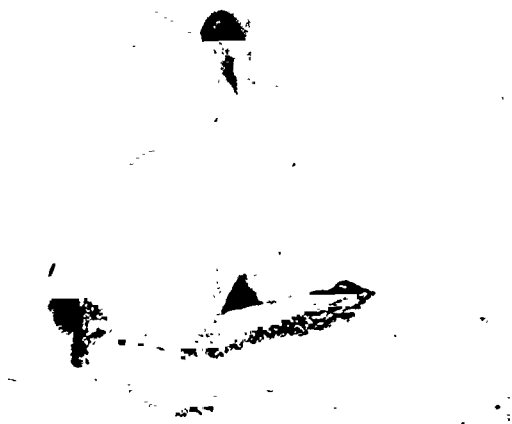
FIG. 218.—Contractures of right hind limb 65 days after ligation of both arteries. Gangrene of digits developed on 70th day.

At post-mortem it was found that in the surviving portion of the limb many muscles had become necrotic in whole or in part. The necrotic areas

FIG. 219.—Microphotograph showing changes at 28th day after arterial ligation. Note part of a necrotic zone surrounded by demarcation zone of young fibrous tissue and atrophied fibres. (× 30.)

were surrounded and replaced by fibrous tissue (*Fig.* 219), which caused shortening and fixation of muscles and thus contractures. Surviving muscle was thin and atrophied and often infiltrated to a slight extent by young

fibrous tissue.   Replacement of necrotic muscle was a very slow process.   No rapidly developing massive fibrosis of muscle was noted.   The degree of fibrosis and the incidence and development of contractures were not materially affected by venous ligation.

Type C.—During life the main feature was marked muscle weakness for as long as four weeks, followed by a slow and finally almost complete recovery of power.   Very little swelling, no induration, and no contractures were noted.   The limb was very thin.   Gangrene appeared in the digits after the skin changes noted above, but remained strictly limited.   After spontaneous amputation, the stump healed.

The animals were killed after several months.   The muscles of the limb were thin.   Necrosis had occurred in only a few muscles and there were corresponding replacement changes.   The other muscles showed the changes of atrophy.

Group II.—The majority of animals in this group died before the twelfth day.   During life the changes were very similar to those in *Type B* of gangrene at the corresponding time.   They might be classified as cases of potential gangrene, and probably most, had they survived, would have developed late peripheral gangrene.   Those surviving beyond the second week developed contractures.

In this group are included also all cases of doubtful early gangrene. There were six in *Series* 3 and five in *Series* 4.

At post-mortem the alterations were similar to but less diffuse than those in the surviving portion of the limb in *Type A* gangrene, and probably they represent an early stage of the changes found later in *Type B* gangrene. The distribution of muscle necrosis was very similar to that in the latter. In the late stage the appearances, apart from gangrene, were identical with those in *Type B* gangrene.

Group III. — All animals in this group died at an early stage before recovery of power was complete.   The probable outcome, so far as could be judged, was a full functional recovery or a late limited gangrene as in *Type C* gangrene.   The structural changes were those of scattered necrosis of muscle.

Group IV.—The ultimate result in this group was an apparently complete recovery of function in the limb after a period of muscle weakness varying from three days to eleven weeks.   In some the limb became thin and atrophied to a degree corresponding roughly to the duration of muscle weakness.   In the early stages swelling was slight and transient, or absent, and subsequently no induration or contractures were noted.

In *Series* 1 recovery occurred in all within a few days.   In *Series* 2 weakness was more prolonged, lasting on an average for eighteen days.   In the other series the average duration of weakness was as follows : in *Series* 3. twenty-four days ; in *Series* 4, thirty-eight days ; and in *Series* 5, forty-seven days.

At post-mortem the changes were atrophy of muscle, and, in some animals, necrosis of muscle affecting usually one or more muscles of the quadriceps group.

DISTRIBUTION OF MUSCLE NECROSIS.

Involvement of the different groups of muscles in cases with scattered muscle necrosis is shown in *Table I.* Cases of very diffuse necrosis are not included.

Table I.—Involvement of Muscle Groups in Cases of Scattered Necrosis of Muscle.

SERIES	LIGATION	TOTAL CASES	QUADRI-CEPS	POSTERIOR THIGH GROUP	CALF GROUP	LEG EXTENSORS
1	External iliac artery	1	1	—	—	—
2	Common iliac artery .. · ..	7	6	1	—	—
3	Both arteries	10	10	6	8	4
4	Both arteries and common iliac vein	13	12	6	9	8
5	Both arteries and inferior vena cava	4	3	3	3	3
6	Both arteries and temporary ligation for 24 hours of common iliac vein	3	3	2	2	1
7	Both arteries and temporary ligation for 24 hours of common iliac vein ..	2	1	1	2	1
	Totals	40	36	19	24	17

The distribution of necrosis was remarkably regular both as regards groups of muscles and individual muscles of each group. For example, the quadriceps group was almost invariably affected and the superficial calf group with great frequency. The distribution of necrosis indicates the distribution of the blood entering the limb. The regularity of distribution of necrosis is evidence that the distribution of blood entering the limb after arterial occlusion is determined by the anatomical situation of the conducting collateral channels. Obviously. moreover, the distribution of blood is altered by arterial occlusion but the proximal muscles are not supplied at the expense of the distal. Necrosis of muscle is a process of infarction of a considerable portion of the muscle and not the focal necrosis which would be expected if. as Brooks thought. the capillaries in certain areas were closed while those in the immediate neighbourhood remained open. Ligation of the vein does not affect the distribution or extent of muscle necrosis.

RESULTS.

The distribution of each series into groups is shown in *Table II.*

The incidence of gangrene is equal in *Series* 3 and 4, and is not appreciably altered if one includes as gangrene those doubtful cases in *Group II* previously mentioned. It is not conspicuously different in *Series* 6 and 7. where temporary venous occlusion was tried. It is *increased* in *Series* 5 by

ligation of the inferior vena cava. The division of the cases of gangrene into the different types is shown in *Table II*. The distribution into types is notably similar in *Series* 3 and 4, while in *Series* 5 the proportion of massive gangrene is higher. In *Type A* accurate determination of the time of appearance of gangrene was not possible, though it can be stated that there was no appreciable difference in the different series. In *Type B* the earliest date of appearance of gangrene was the twenty-first day and the latest the seventieth day. The average time of appearance of this type of gangrene was

Table II.—Results of Vascular Ligations.

SERIES	LIGATION	TOTAL CASES	GROUP I Gangrene	GROUP II Severe Disturbances	GROUP III Moderate Disturbance	GROUP IV Slight Disturbances	DEATH BEFORE 10TH DAY	TYPES OF GANGRENE A	B	C
1	External iliac artery	10	0	0	0	10 = 100%	0	—	—	—
2	Common iliac artery	10	1 = 10%	0	0	9 = 90%	1 = 10%	1	—	—
3	External and common iliac arteries	30	13 = 43%	8 = 27%	2 = 7%	7 = 23%	11 = 37%	4	9	—
4	Both arteries and common iliac vein	30	13 = 43%	10 = 34%	3 = 10%	4 = 13%	8 = 27%	3	8	2
5	Both arteries and inferior vena cava	10	6 = 60%	2 = 20%	0	2 = 20%	3 = 30%	4	1	1
6	Both arteries and temporary ligation of common iliac vein for 24 hours	11	5 = 45%	2 = 18%	0	4 = 37%	1 = 9 %	2	3	—
7	Both arteries and temporary ligation of inferior vena cava for 24 hours	8	4 = 50%	0	1 = 12%	3 = 38%	3 = 30%	2	2	—

identical in *Series* 3 and 4, namely the thirty-fifth day. In *Type C* gangrene appeared in one case during the sixth week and in the others during the eleventh week.

The incidence of full recovery is shown in *Table II* by the figures for *Group IV*. The difference between *Series* 3 and 4 is perhaps scarcely decisive, but the average duration of muscle weakness in the different series suggests strongly that ligation of the vein delays the return of power. Ligation of the inferior vena cava delays functional recovery still further.

The mortality during the first ten days after ligation is shown in *Table II*. The figures provide evidence of a possible benefit from venous ligation, since the mortality is higher after arterial ligation alone. Similar but more striking results were obtained by Brooks and Martin. The cause of death after ligation of large arteries will not be discussed in this paper.

THE IMMEDIATE RESULTS OF OCCLUSION OF THE MAIN VESSELS OF A LIMB.

The immediate effects which have been studied are those related to the intravascular pressure, the mass of blood in the limb, the volume flow of blood per minute, and the oxygen consumption of the tissues of the limb per minute. For these experiments dogs were used. The animals were anæsthetized by intravenous injection of sodium barbital, 250 mgrm. per kilo. of body weight. In most instances heparin was injected to prevent clotting of blood. The amount was 30 mgrm. per kilo. of body weight.

INTRAVASCULAR PRESSURE.

The vessels occluded were those of one hind limb. Continuous records were taken of pressure in the carotid artery and of the limb arterial and limb venous pressures below the level of occlusion. The limb pressures were recorded from cannulas tied into a side branch of each main vessel or from

Table III.—RESULTS OF VENOUS OCCLUSION WITHOUT ARTERIAL OCCLUSION.

Carotid pressure ——— Limb arterial ...... Limb venous —.—.—.—
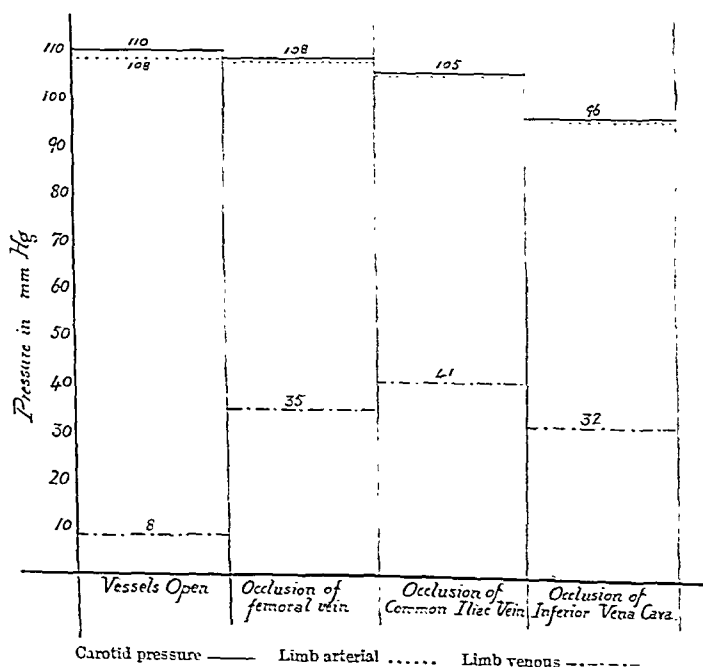
T-cannulas in the main vessels. Figures for arterial pressure signify mean arterial pressure in mm. Hg.

1. **Venous Occlusion without Arterial Occlusion.** — The femoral vein, iliac vein, and the inferior vena cava were occluded in turn. The average results of six experiments are shown in *Table III*.

Points.—

a. The limb arterial pressure follows closely the carotid pressure.

b. Occlusion of the femoral vein is without effect on the limb arterial pressure. Occlusion of the iliac vein or of the inferior vena cava causes a fall in pressure in the carotid and limb arteries. The fall is due to a lessened return of blood to the heart.

2. **Arterial Occlusion.**—The changes in the limb pressures and the carotid pressure after occlusion of the main artery were measured. The results of fifteen experiments are shown in *Table IV*. The figures refer to mean pressures in mm. Hg. at ten minutes after occlusion.

Table IV.—Changes in Limb Pressure and Carotid Pressure after Arterial and Venous Occlusion.

Femoral Vessels

EXPERI-MENT No.	CAROTID PRESSURE	ARTERIAL OCCLUSION		RISE AFTER VENOUS OCCLUSION	
		L.A.P.	L.V.P.	L.A.P.	L.V.P.
10	125	62	—	16	—
24	90	48	—	7	—
25	110	78	4	2	17
26	110	64	4	0	20
33	150	106	6	9	26
34	144	78	4	16	18
35	122	78	4	0	6
40	90	42	4	0	3
Average	118	69	4·2	6·2	15

Iliac Vessels

EXPERI-MENT No.	CAROTID PRESSURE	ARTERIAL OCCLUSION		RISE AFTER VENOUS OCCLUSION	
		L.A.P.	L.V.P.	L.A.P.	L.V.P.
6	112	58	—	3	—
8	120	84	—	0	—
13	136	53	—	7	—
16	112	16	4	28	30
19	120	36	5	5	10
20	122	76	5	0	4
38	130	57	6	19	24
Average	126	54	5	9	17

L.A.P. = Limb arterial pressure in mm. Hg.   L.V.P. = Limb venous pressure in mm. Hg.

Points.—

a. The effect on the mean carotid pressure is insignificant.

b. The limb arterial pressure falls sharply, and then, recovering quickly, assumes a level (*Fig. 220*). If a high level is reached, pulsations return. The level varies greatly in different animals.

3. **Occlusion of the Accompanying Main Vein after Occlusion of the Main Artery.**—The results of fifteen experiments are shown in *Table IV*.

Points.—

a. In 50 per cent of experiments the effect of venous occlusion on the limb arterial pressure is negligible. In the remainder a rise of pressure occurred. The rise is therefore inconstant.

b. The rise in venous pressure is always greater than any simultaneous rise in pressure in the arteries. The same observation was made by Brooks and Martin.

c. Occlusion of the main vein has a local action. whereby it raises the pressure in the veins and sometimes in the arteries of the limb, and a more general action, whereby it lessens the return of blood to the heart and may thus cause a fall in the general systemic pressure.
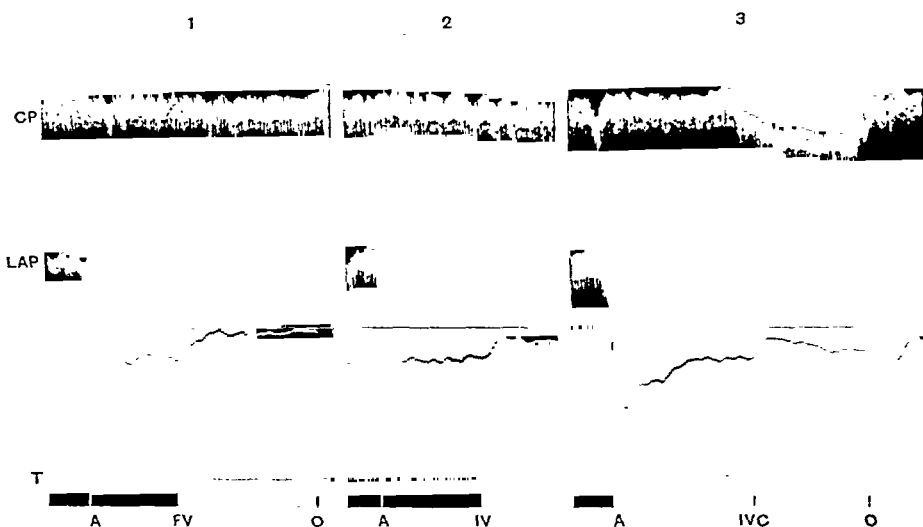
FIG. 220.—Experiment 46. Successive occlusion of femoral artery and main veins at different levels. (1) Occlusion of femoral vein at FV; (2) Occlusion of iliac vein at IV; (3) Occlusion of inferior vena cava at IVC. At A, occlusion of femoral artery; at O, release of vein. Note decrease of rise from 1 to 3 CP, Carotid pressure ; LAP, Femoral artery pressure : T, Time marker in 5 seconds.

4. Occlusion of the Vein at a Level Higher than that of Arterial Occlusion.— The effect of such occlusion was tested in six experiments. In three experiments the results were the reverse of those previously reported (*Fig.* 220). In the others the increased rise was found. The phenomenon is therefore not constant.

An important fact was noted. On two occasions occlusion of the inferior vena cava caused a marked fall in general arterial pressure, during which the limb arterial pressure fell to below the level it had attained after arterial occlusion alone. In such instances the effect of occluding the vein on the general systemic pressure outweighs the local effect on the intravascular pressure in the limb.

5. Mechanism of Rise of Pressure.—The theory of Leriche and Fontaine was tested with the following results :—

a. The rise of arterial limb pressure, if present initially, occurs after

denervation of the limb and removal of the entire lumbar sympathetic chain.

b. No change in the volume of a limb occurs during occlusion of the main vessels of the opposite limb, if the carotid pressure is unaffected.

c. During occlusion of the main artery no rise of limb arterial pressure occurs on occlusion of the contralateral main vein.

It may be concluded that the rise of pressure is independent of the integrity of the nerves of the limb. There was no evidence of a sympathetic reflex action involving the vessels either of the same or of the opposite limb.

The reaction of the vessels of the limb to occlusion of the main artery and vein must also be considered.

In order to determine the influence on the tone of the vessels of occlusion of the main vein with occlusion of the main artery, the following experiment was devised. The main artery and vein and the larger nerves of the limb were isolated in the upper part of the thigh. All other structures were severed, including the bone, so that blood entered and left the limb only by the main vessels. The limb pressures were recorded. On occlusion of the artery the pressure in arteries and veins reached a level of about 5 mm. Hg. in three to four minutes. After simultaneous occlusion of artery and vein the same result was obtained in about five to six minutes, the venous pressure rising for a brief period as the arterial pressure began to reach a level. Division of the nerves did not affect the result.

Thus occlusion of the vein does not prevent the rapid loss of vascular tone which follows complete arterial occlusion. That its effect on vascular tone is different in lesser degrees of arterial obstruction is certainly unlikely. More probably it accentuates the active vasodilatation on the arterial side. A similar experiment on the vascular tone in man has been described by Kendrew.[8]

6. **Duration of the Rise of Limb Pressure.**— In four experiments the artery and vein of one limb and the artery of the opposite limb were divided between ligatures. The pressures in artery and vein of each limb were measured after twelve hours in two experiments and after twenty-four hours in two. It was found that the rise of pressure in the limb arteries after venous occlusion did not last for more than twelve hours.

MASS OF BLOOD IN THE LIMB.

Plethysmographic tracings showed that the volume of the limb fell after arterial occlusion and then, on subsequent venous occlusion, rose to a level well above the normal. The increased volume affected the most distal parts, as shown by a record of foot volume, and its duration was variable. The same changes occurred after denervation but were less marked, since the vessels were initially relaxed.

The quantity of blood in the limb is, therefore, greater than normal for a variable period after occlusion of both vessels. It follows that there must be considerable increased filling of vessels, most probably on the venous side, and a marked slowing of the flow of blood.

VOLUME FLOW OF BLOOD PER MINUTE, OXYGEN UTILIZATION, AND OXYGEN CONSUMPTION OF THE TISSUES PER MINUTE.

The experiments in this section were carried out in conjunction with Dr. M. L. Montgomery. The *Stromuhr* designed and described by him was used for the measurement of volume flow. A brief consideration of his experiments, already quoted, is required here, and the experimental conditions must be clearly understood. The flow which was measured includes the greater part of the blood going to the posterior extremity. The blood enters the branches of the main vessel above the obstruction on the femoral artery. A part passes through the capillary bed of the tissues which these branches normally supply. The remainder enters the limb below the obstruction through collateral channels. The tissues of the whole limb may be divided into the part above the obstruction and the part below, i.e., into 'unobstructed' and 'obstructed' portions of the limb. The effect of proximal venous ligation at least is to diminish the total flow through the whole limb.

We are concerned, however, mainly with the flow through the obstructed portion, and, unfortunately, there is as yet no accurate method of measuring it. Nevertheless, there is much indirect evidence of a decreased flow through the obstructed portion after venous ligation, and it may be assumed that venous ligation which, as Montgomery showed, causes a diminution of the total volume flow, produces also a corresponding decrease in the flow through the obstructed portion.

If one accepts the hypothesis that the volume flow through the obstructed portion varies directly with the total volume flow, one can obtain an estimate of the oxygen consumption of the tissues of the obstructed portion per minute. To do so the total volume flow is measured and also the oxygen utilization (i.e., the difference in oxygen content of the arterial and venous blood) below the level of obstruction. The product of volume flow per minute and oxygen utilization gives an index of the oxygen consumption of tissues per minute.

Oxygen Utilization. — The following technique was adopted. From T-cannulas in iliac and femoral vessels samples of blood were withdrawn under oil before occlusion, during occlusion of the femoral artery, and during occlusion of artery and vein. The oxygen content of each sample was measured in a Van-Slyke-Neil apparatus. Blood from the iliac vein may be considered as blood from the tissues of the whole limb, that from the femoral vein as from the tissues of the obstructed part. A typical example is shown in *Table V*.

Results.—

a. The arterial oxygen content remained constant above and below the obstruction. Therefore the collateral channels are larger than capillaries.

b. The oxygen utilization and the co-efficient of utilization rose in both iliac and femoral blood after arterial occlusion. The rise was higher below the occlusion.

c. On subsequent venous occlusion there was a further rise in utilization and co-efficient of utilization, and the rise was more marked below the obstruction.

These results indicate an increased stasis after venous occlusion.

Table V.—Oxygen Utilization.

		ILIAC ARTERY O₂ Content	ILIAC VEIN O₂ Content	ILIAC VESSELS O₂ Utilization	ILIAC VESSELS Coeff. of Utilization	FEMORAL ARTERY O₂ Content	FEMORAL VEIN O₂ Content	FEMORAL VESSELS O₂ Utilization	FEMORAL VESSELS Coeff. of Utilization
1	Vessels unoccluded	Vols. % 20·04	Vols. % 15·45	Vols. % 4·59	0·23	Vols. % 20·04	Vols. % 12·63	Vols. % 7·41	0·37
2	Femoral artery occluded ..	20·04	13·01	7·03	0·35	20·04	9·91	10·13	0·51
3	Femoral artery and vein occluded ..	20·10	10·95	9·15	0·46	20·10	7·84	12·26	0·61

Oxygen Consumption of the Tissues per Minute.—

a. The Obstructed Portion.—The oxygen consumption of the tissues of the obstructed portion of the limb was estimated as the product of the volume flow through the iliac artery and of the oxygen utilization of blood in the femoral vessels. The consumption was measured with the vessels unoccluded, the femoral artery occluded, and the femoral artery and vein occluded. The occlusion was repeated while recording the pressures in the femoral vessels. *Tables VI* and *VII* are typical examples, and a record is shown in *Fig. 221.*

Table VI.—Volume Flow and Limb Pressures.

VESSELS	CAROTID PRESSURE IN MM. HG.	L.A.P.	L.V.P.	VOL. FLOW PER MIN.
No occlusion	138	128	6	c.c. 95·60
Occlusion femoral vein ..	139	129	86	64·40
Occlusion femoral artery ..	144	78	3·8	30·46
Occlusion femoral artery and vein	144	94	22	25·81
No occlusion	142	128	4·5	87·26

L.A.P. = Limb arterial pressure in mm. Hg.   L.V.P. = Limb venous pressure in mm. Hg.

Table VII.—Estimated Oxygen Consumption of Limb.

VESSELS	CAROTID PRESSURE IN MM. HG.	VOL. FLOW PER MIN.	A—V.O₂ DIFF. IN FEMORAL VESSELS	COEFF. OF UTILIZATION	ESTIMATED O₂ CONSUMPTION PER MIN.
No occlusion	108	c.c. 74·03	Vols. % 8·50	0·38	c.c. 6·20
Occlusion femoral artery	100	28·60	12·64	0·57	3·62
Occlusion femoral artery and vein ..	98	22·40	15·26	0·68	3·42
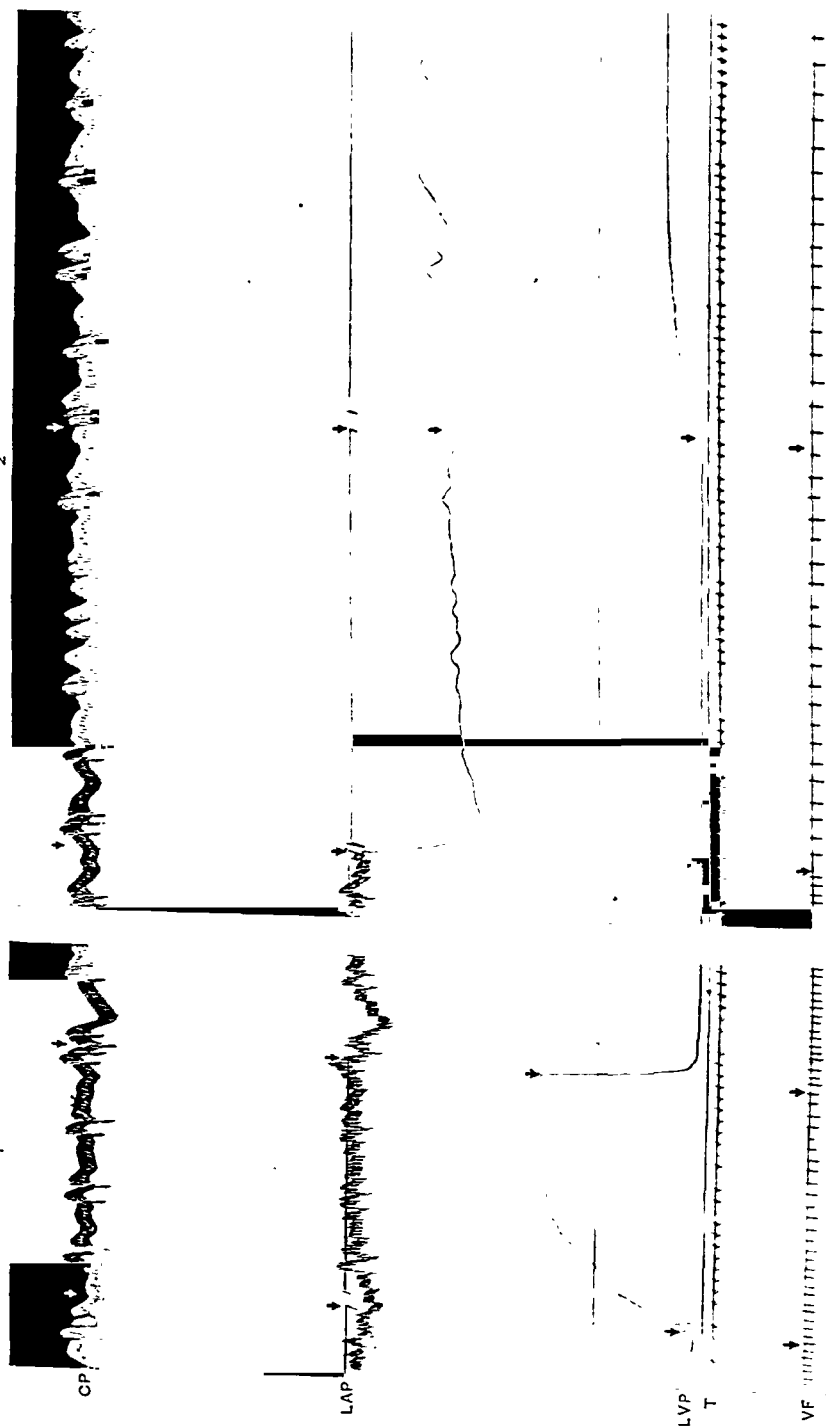
Fig. 221.—Experiment 34. Successive occlusion of femoral vessels. Effect on volume flow through iliac artery per minute, carotid and limb pressures. C—o, Occlusion of femoral vein alone : flow 61·16 c.c. per min. A—v, Occlusion of femoral artery alone : flow 30·16 c.c. per min. After v, Occlusion of femoral artery and vein : flow 25·81 c.c. per min. CP, Carotid pressure ; LAP, Femoral artery pressure ; LVP, Femoral vein pressure ; VF, Record of volume flow ; T, Time-marker in 5 seconds.

The volume flow falls after occlusion of the artery and still further after subsequent occlusion of the vein. The oxygen utilization rises, but the oxygen consumption per minute as calculated by this method decreases after venous occlusion.

b. The Whole Limb.—The oxygen consumption was measured as the product of volume flow through the iliac artery and of the oxygen utilization in the iliac vessels. The results were similar to those in the previous experiments.

SUMMARY OF IMMEDIATE EFFECTS OF OCCLUDING THE MAIN ARTERY AND MAIN VEIN OF A LIMB.

1. *After Occlusion of the Main Artery of a Limb.*—

a. The limb pressures, arterial and venous, fall.

b. The mass of blood in the limb and the volume flow of blood per minute are diminished.

c. The oxygen utilization, and co-efficient of utilization, of blood in the main vessels above and below the occlusion are increased.

d. The oxygen consumption per minute of the tissues of the whole limb and of the tissues below the obstruction is diminished.

2. *On Subsequent Occlusion of the Main Vein at the Same Level.*—

a. The limb pressures may increase, but the rise in limb arterial pressure is inconstant and frequently insignificant, and probably in any case lasts for no more than twelve hours. The rise of arterial pressure does not depend upon the integrity of the nerves to the limb or of the lumbar sympathetic system. The rise is probably purely a mechanical effect of obstruction to the venous outflow. There is no evidence of increased tone in the vessels. The rise in limb venous pressure is always greater than the corresponding rise in limb arterial pressure.

b. The mass of blood in the limb becomes greater than normal.

c. The volume flow of blood per minute is further diminished through the whole limb, and probably through the tissues below the obstruction.

d. The oxygen utilization and coefficient of utilization of the blood in the main vessels above and below the occlusion are further increased.

e. In spite of increased oxygen utilization, the oxygen consumption per minute of the tissues of the whole limb and of the part below the occlusion is still further diminished.

3. *Occlusion of the Vein at a Higher Level as Compared with Occlusion at the same Level as Arterial Occlusion.*—

a. The limb arterial pressure may rise still higher. The rise is inconstant and the result may be a fall associated with a dangerous fall in general arterial pressure when the lower end of the inferior vena cava is occluded.

b. The reduction in volume flow per minute is more marked and always considerable.

DISCUSSION.

The results of the survival experiments indicate definitely that ligation of the main vein does not lessen the incidence of gangrene which follows ligation of the main artery of a limb. Venous ligation does not materially

affect the nature, extent, or time of appearance of subsequent gangrene, and probably lessens the chances of complete recovery of function. It delays the return of power to the muscles of the limb. The only apparently beneficial effect of obstructing the veins was a reduction in the immediate mortality following occlusion of large arterial channels. Temporary occlusion of the main vein for twenty-four hours confers no certain benefit.

The main experimental basis for the belief that ligation of the vein decreases the incidence of gangrene is the work of Brooks and Martin. This investigation failed to confirm their work in a larger series of experiments, in which the animals have been kept alive for longer periods, and in which special effort has been made to exclude fallacies in technical execution of the operations, and in interpretation of gangrene.

The claim put forward by Holman and Edwards, that ligation of the vein at a higher level still further diminishes the incidence of gangrene, is based on experiments which are open to objection on two points. Firstly, they employed no control series of ligation of artery alone, but used the figures quoted by Brooks and Martin for comparison with their results. Secondly, they did not in all experiments employ the arterial ligation of Brooks and Martin. In 18 experiments they reported gangrene in 11 per cent of cases when the common iliac artery and inferior vena cava were ligated. In the present *Series* 2 ligation of the common iliac artery alone gave gangrene in only 10 per cent of cases. The results in the present *Series* 5, which forms a repetition of the remainder of their experiments, are completely at variance with theirs.

In view of the results of the survival experiments it might seem unnecessary to review the theories of the supposed beneficial action. These theories, however, have become to some extent current belief, and are based on experimental results which are of considerable theoretical importance.

The theory of 'restoration of the balance of the circulation' is an example of vague phraseology. There occurs after venous occlusion a further diminution of the volume flow of blood per minute and of the rate of flow. Such conditions can scarcely be designated a restoration of circulatory balance.

Venous ligation does not alter the incidence of gangrene or the severity or distribution of muscle necrosis. Therefore it does not effect a dispersion of the arterial blood to the distal parts. For the same reasons Brooks' theory of a more homogeneous distribution of arterial blood is not acceptable.

The belief that ligation of the vein aids by keeping the blood in longer contact with the tissues cannot at present be countenanced. The oxygen consumption per minute of the tissues of the limb as a whole, and of the obstructed portion, is lessened by venous ligation, although the limb contains a quantity of blood greater than normal and the oxygen utilization of the blood is increased. Volume flow per minute is apparently the most important factor for oxygen consumption.

The observation that for some time after venous ligation the calibre of the arterial vessels is increased has given rise to the theory of a 'richer collateral bed'. The observation is very probably correct, but has no great bearing on the question of nutrition of tissues or gangrene.

It is not agreed that venous occlusion acts as a 'masked sympathectomy'. The initial rise of limb arterial pressure may be only temporary, and an active dilatation of vessels on the arterial side is possible, and even probable. The reactions of the vessels to occlusion, however, are independent of the integrity of the nerves to the limb.

The dangers of marked venous obstruction in a limb are well known. In clinical cases gangrene has been reported from venous obstruction without arterial obstruction.[1, 6] Thus temporary occlusion would seem preferable to permanent occlusion of the vein. Moreover, it is usually stated that the supposed benefits accrue during an early period. However, no beneficial action of temporary venous occlusion for twenty-four hours has been found in this work.

The experiments on proximal venous ligation have demonstrated that this procedure has effects which are decidedly deleterious.

Special Features.—Two features merit further consideration—namely, the special vulnerability of tissues to ischæmia, and the occurrence of contractures.

Experiments by Brooks[2] showed that with a permanent severe deprivation of arterial blood the skin of a limb was more extensively affected than the muscles, while, if the deprivation were temporary, the changes were more severe and extensive in the muscles. He concluded that skin is less tolerant than muscle of severe permanent diminution of blood-supply, but survives better a temporary deprivation of blood. The evidence is not altogether satisfactory, since, in his experiments, the distribution of the blood to the different tissues and the degree of ischæmia could not be controlled. On a priori grounds such tissue characteristics are improbable, and, moreover, the survival experiments reported here do not favour such a belief. In rabbits, if the death of skin were extensive, there was invariably diffuse necrosis of the muscles in the portion covered by surviving skin. In all cases of peripheral gangrene, and in some cases without death of skin, there was also scattered necrosis of muscles. The assumption is reasonable that muscle, as a more highly differentiated tissue than skin, withstands ischæmia less well. Massive death of skin to a higher level than that of subjacent structures can be more satisfactorily explained by the distribution of blood to the tissues, the deeper structures being more generously supplied than the superficial, at least below the knee.

The occurrence of contractures after arterial ligation alone has not previously received attention. Brooks[2, 4] has shown in an admirable series of experiments that the massive fibrosis of muscle which produces rapid development of contractures, such as Volkmann's contracture, is a result not of ischæmia but of a complete obstruction of the venous return from the muscles. His experiments have been confirmed by Middleton,[13] who has demonstrated that the sternomastoid tumour of infants, which gives rise to torticollis, has a similar origin. There is at present a tendency towards the total exclusion of arterial obstruction from the causation of contractures. In the survival experiments on arterial occlusion all animals which did not develop massive gangrene or make a full or nearly full functional recovery, and which survived beyond the second week, developed contractures of the limb. There were ten cases in all. Admittedly peripheral gangrene of the

digits ultimately appeared in all except one, but usually contractures were well established before the appearance of gangrene. The contractures were due to the development of fibrous tissue which slowly replaced the necrotic muscle. The fascia also shared in the fibrotic process. The slow rate of fibrous replacement was no doubt due to the diminished blood-supply to the limb.

It is not suggested that arterial obstruction necessarily plays a part in such conditions as Volkmann's contracture. Obstruction of the main artery of a limb may, however, produce a slowly developing contracture of the limb. Simultaneous ligation of the main vein apparently has no influence on the development of contracture.

CONCLUSIONS.

1. The immediate and late effects of ligating the main artery and main vein of a limb have been studied experimentally.

2. The results of the investigation are not in accord with previous experimental work on the subject. They do not support the current belief that ligation of the main vein diminishes the incidence of gangrene which follows ligation of the main artery.

3. Ligation of the vein at a higher level than arterial ligation *increases* the incidence and extent of tissue death.

4. Theories previously held to account for the supposed benefits of venous ligation have not withstood experimental trial.

I wish to express my thanks to Dr. D. B. Phemister, Professor of Surgery, University of Chicago, for his interest and advice during this investigation.

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ON THE INNERVATION OF THE BLOOD-VESSELS OF THE UPPER EXTREMITY: SOME ANATOMICAL CONSIDERATIONS.

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THE wide adoption of the operation of sympathectomy for vascular lesions has necessitated a more exact knowledge of the anatomy of the sympathetic cord, in particular in the cervical and lumbar regions. The innervation of the blood-vessels to the lower limb has recently been carefully investigated by Woollard,[40] and the operation of lumbar sympathectomy for conditions of arterial spasm in the lower extremities is invariably completely successful, provided that no extensive obliterative changes have superseded the spasm in the arterial wall. Cervico-dorsal sympathectomy for Raynaud's disease, however, although resulting in a definite improvement in the condition of the upper limb, rarely gives a complete recovery. Only too frequently recurrence of the symptoms appears in one or more fingers, usually but not invariably on the ulnar side of the hand. This incomplete result, in marked contrast to that in the lower extremity, has been noted by many surgeons (Davis and Kanavel,[5] Adson and Brown,[1] Telford and Stopford,[36] Royle,[30] Fulton,[9] Hesse[11]), and varied explanations have been advanced to account for this apparent anomaly.

In 1912 Todd[37] described in detail the contribution from the second thoracic nerve to the brachial plexus, and he showed that this communicating branch consisted of both spinal and sympathetic fibres. Later[38] he emphasized its clinical importance in vascular changes produced by pressure on the lowest trunk of the brachial plexus. Kuntz[17] has explained the imperfect results of cervico-dorsal sympathectomy by observing that in a large percentage of cases this intrathoracic ramus arises from the second thoracic nerve and joins the first usually proximal to the origin of the first intercostal nerve. This constitutes a pathway through which sympathetic fibres may leave the sympathetic trunk below the first thoracic ganglion and enter the brachial plexus. Following this suggestion Adson and Brown[2] have operated on a case by the dorsal route with complete extirpation of the stellate ganglion and of the thoracic sympathetic cord as far distal as to include the second thoracic ganglion. They report: "The results obtained in the upper extremities by this procedure are comparable to those obtained with the removal of the lumbar sympathetic ganglia in the lower extremities." It would seem, therefore, that cervico-dorsal ganglionectomy does not often give a complete sympathetic denervation of the hand unless the second thoracic ganglion or its corresponding rami are also included.

Lewis,[26] however, rightly suggests that there can be comparatively few

fibres passing by this channel to the brachial plexus, and he considers that it is most unlikely that spasm of the vessels to the extent of obliteration in all the fingers could ever occur as a response to nerve impulses passing exclusively along this path. From the experimental researches that he has carried out with his co-workers, he comes to the conclusion that sympathectomy will fail to cure the symptoms however wide the excision of nerves, because the real cause of the spasm is peripheral and not central, e.g., a local fault of the vessels. He describes with Landis[27] a case of severe Raynaud's disease of long duration on which right cervico-dorsal sympathectomy was performed. The main trunk of the sympathetic was cut below the second thoracic ganglion and the corresponding rami divided ; all nerves joining the inferior cervical ganglion were defined and divided, and the ganglion together with the second thoracic ganglion and the intervening portion of the sympathetic trunk removed in one piece. Following the excision the fingers on the operated side were much warmer than on the opposite side and showed a decidedly lessened tendency to lose their circulation. " It is manifest, however, that the primary cause of spasm remains unchanged in the right hand, and that it does not consist of vasomotor impulses, none of which can now reach the vessels of this hand." Immersion in water at 15° C. produced complete loss of circulation in parts of both hands, though the area involved was less in the right hand than in the left. The relief of the symptoms by sympathectomy is due, according to Lewis, not to removal of abnormal vasomotor impulses, but to removal of normal ones which, even in the normal person on exposure of the body to a cold atmosphere, result in partial vasoconstriction. In Raynaud's disease the closure of the digital vessels is complete. To explain this, Lewis postulates a local fault, which according to him may be of two kinds : (1) Structural changes in the arteries such as intimal thickening ; or (2) A more powerful response of these vessels to such a local stimulus as cold.

Lewis's hypothesis does not, however, allow for the extraordinarily perfect results of lumbar sympathectomy for conditions of vascular spasm in the lower limbs. Raynaud's disease is manifestly a diffuse condition ; and if a local fault in the digital vessels of the fingers is postulated, why not also for the toes ? Furthermore, the intimal thickening or pathological changes in the vessel wall may be regarded, as Lewis admits, as consecutive to the vascular spasm and a diminished blood-flow. Simpson, Brown, and Adson[32] have pointed out that Lewis's evidence is based on cases of advanced degrees of Raynaud's disease in which pathological changes in the vessels had been superadded to the spasm, and therefore is not applicable to the more usual mild cases. They advance evidence in support of the neurogenic origin of the condition. In severe and complicated cases where there are additional vascular changes, even after interruption of all sympathetic fibres, it is admittedly possible for the arteries of certain digits to enter into spasm with resulting cyanosis. It is concluded that in these severe complicated cases there is an abnormal response of the arteries, which is most striking in the fingers that have undergone ulceration of the tip, but this is attributed to *secondary* changes that have taken place in the arteries and arterioles. This point seems of some importance. In the clinical report of the case quoted above

Lewis remarks that "small scales of dry gangrene often occurred on her finger tips."

A third view has been advanced in regard to the frequent imperfect results of sympathectomy. Leriche, who still advocates peri-arterial sympathectomy with good results in simple uncomplicated conditions of vascular spasm,[24] has with Fontaine[25] published some recent deductions from his review of the results of operations on the sympathetic nervous system, whether ganglionectomy, ramisection, or peri-arterial sympathectomy. Such operations are all followed by an active vasodilatation, more or less lasting. This makes itself felt especially distally, but one can find traces of it proximally and often on the opposite side. These results, they hold, can be readily explained if one advances the hypothesis of sensory fibres in the vessels. On this assumption they postulate vascular reflexes, of which they recognize : (1) Peripheral vascular reflexes having their centre in the intramural plexuses ; (2) Vascular changes through axone reflexes ; (3) Intrasympathetic reflexes which have their centre in the ganglia of the sympathetic trunk ; (4) Medullary vascular reflexes ; (5) Cerebral vascular reflexes. The vascular changes, write Leriche and Fontaine, are the same after either peri-arterial sympathectomy or operations upon the sympathetic trunk, but in the latter case the modifications in the local circulation are more marked as more pressor fibres have been cut. "There is a quantitative but not a qualitative difference between these two operations."

It is clear that in the operation of cervico-thoracic sympathectomy many post-ganglionic fibres from cells situated in the middle cervical ganglion must remain intact. Kerper,[16] in his experimental work on dogs, found that following excision of the stellate ganglion there remained a number of nerve-fibres in the adventitia, and some in the media. Those in the superficial layers of the adventitia were mainly myelinated, those in the deeper layers of the adventitia and the media were mainly unmyelinated. Kuntz[18 19] believes that at least some of these fibres are sensory in function. Many of the unmyelinated may possibly have been post-ganglionic fibres from the middle cervical ganglion. The assumption of purely sensory fibres to blood-vessels and the possibility of local vaso-reflexes is not new. It has been expressed by many clinical workers in this field, notably Läwen[23] and Friedrich.[8] The evidence has recently been critically reviewed by Stopford.[34] There is no clear anatomical proof of any afferent path from the blood-vessels of the limbs to the ganglionated sympathetic cord. Thus up to the present Leriche's hypothesis has received no definite support from anatomical research, though there is some suggestive evidence in its favour.

Lastly, in explanation of the incomplete results of cervicodorsal sympathectomy, we have the possibility of persistence of the peri-arterial nervous plexus arising from the sympathetic cord close to the origin of the subclavian artery. We do not yet know how far the peri-arterial plexus from the proximal source extends down the upper limbs, or whether there is any marked overlap with the distal source of supply. Woollard[40] has shown in the lower extremity that the overlap is not very great and occurs about the level of the bifurcation of the femoral. In the upper limb it is generally accepted, though unproven, that the proximal source does not reach farther

than half way down the upper arm. Langley[21] and Wiedhopf[39] have found no evidence for the existence of long vasoconstrictor pathways extending along the perivascular tissues as far as the hand, and Dennig[6] also considers that there is none for the vasodilator fibres. It must be admitted that the experimental evidence advanced by these workers against the existence of long perivascular nerve pathways is not in any way conclusive. As regards vasodilator nerves, it seems important to call attention to the work of Kuré and his collaborators from Tokyo[20] who have in recent years suggested a possible new anatomical pathway for vasodilator impulses to blood-vessels through what they term the 'spinal parasympathetic system'. This work is still in progress and awaits further confirmation.

To summarize, we can tabulate the possible factors underlying the incomplete results of cervico-dorsal sympathectomy :—

1. Failure to produce a total sympathetic denervation of the limb, usually from incomplete extirpation of the upper thoracic sympathetic cord.

2. Pathological changes in the peripheral vessels possibly secondary to the vascular spasm, as seems to occur in severe and complicated cases.

3. The possibility of the middle cervical and vertebral ganglia acting as reflex centres : the post-ganglionic fibres from these cell stations remaining intact after cervico-dorsal sympathectomy. This theory necessitates the postulation of afferent vascular nerves.

4. Extension of the peri-arterial nervous plexus as far down as the hand. It will be shown later how in the case of the upper limb the perivascular plexus may not completely degenerate after removal of the stellate ganglion. Whether this has any significance remains to be seen.

All the collected evidence indicates that there is still much to be learnt about the possible anatomical pathways by which nerve impulses can reach the vessels in question, in particular in regard to the upper extremity. It is generally accepted that there are two possible paths for vasomotor impulses to the limbs. " First, a proximal supply, which comes from the lower part of the cervical portion of the sympathetic ganglionated cord and accompanies the subclavian artery in the case of the arm ; and an extension of the aortic sympathetic plexus along the common iliac, external iliac, and femoral arteries in the leg. Secondly, a more distal supply consisting of fine filaments which arise from neighbouring peripheral nerves, and reach the vessels at intervals " (Stopford[34]).

The distribution of the arterial branches of the peripheral nerves in the upper extremity has been carefully studied by Goering,[10] Frey,[7] Kramer and Todd,[15] Hirsch,[12] and more recently by Coates.[4] Anatomical studies directed particularly to the proximal source of vasomotor fibres are singularly lacking, though there are many accounts of the lower cervical sympathetic cord to be found in the works of Potts,[29] Siwe,[33] Jonnesco,[14] Laubmann,[22] Sozon-Jarosevic.[35] and in particular in the excellent and detailed descriptions given by Axford[3] and Hovelacque.[13] Little of anatomical importance can be added to these exhaustive studies, but it is the purpose of this paper to emphasize the significant difference in the anatomical arrangement of the proximal sympathetic supply to the blood-vessels of the upper and lower limbs and to stress certain important surgical relations in regard to the former. A

reinvestigation of the innervation of the blood-vessels of the limbs, based on experimental evidence, is being undertaken. The present investigation has been carried out at the suggestion of Professor J. S. B. Stopford. The lower cervical and upper thoracic sympathetic cord has been carefully dissected on twenty-five sides, attention being particularly directed to the rami communicantes to the roots of the brachial plexus, and to the relationship of nerve-fibres to the subclavian artery.

The most striking feature that became evident in this study has been the great complexity and variability of the branches of distribution from the sympathetic cord and ganglia. A glance at the accompanying sketches (*Figs.* 222–227) will indicate the problem that faces the surgeon in any attempt to produce sympathetic denervation of the upper limb. Some of the rami communicantes arise from the anterior and posterior aspect of the ganglia

FIG. 222.—Right side of body. Showing the distance between the first thoracic ramus and the inferior cervical ganglion. Distance B–B' = 1 in. Note the relation of the superior intercostal artery and vein to the sympathetic cord; the vein lies medial to the artery.

and run for a course of less than $\frac{1}{2}$ cm., a course often entirely hidden from view. Again, the number and exact site of origin of these branches is variable and their relationship to the superior intercostal artery very intimate. The distinction drawn by Axford between white and grey rami is not at all so clear at operation as one would be led to expect from anatomical dissections. It seems evident that in order to ensure complete sympathetic denervation of the upper limb, the operation of ganglionectomy (inferior cervical and the upper two thoracic) is a much surer and more practical proposition than any attempt at cervico-thoracic ramisection.

A second point of some surgical importance was observed soon after the commencement of this study. The contribution of the second thoracic nerve to the brachial plexus, and the existence through this communication of a pathway by which sympathetic fibres can reach the upper limb from well below the inferior cervical ganglion, has been emphasized by Kuntz. But

this is not the only possible loophole in an incomplete cervico-dorsalectomy. The grey rami to the first thoracic nerve frequently arise some distance below the inferior cervical ganglion, even in those cases where the latter is partially or completely fused with the first thoracic ganglion. *Fig.* 222 demonstrates this point very clearly, the distance separating the lower end of the inferior cervical ganglion and the rami communicantes to the first thoracic nerve being as much as one inch. In such cases this is an additional and very important source of sympathetic fibres to the upper extremity. It is essen-
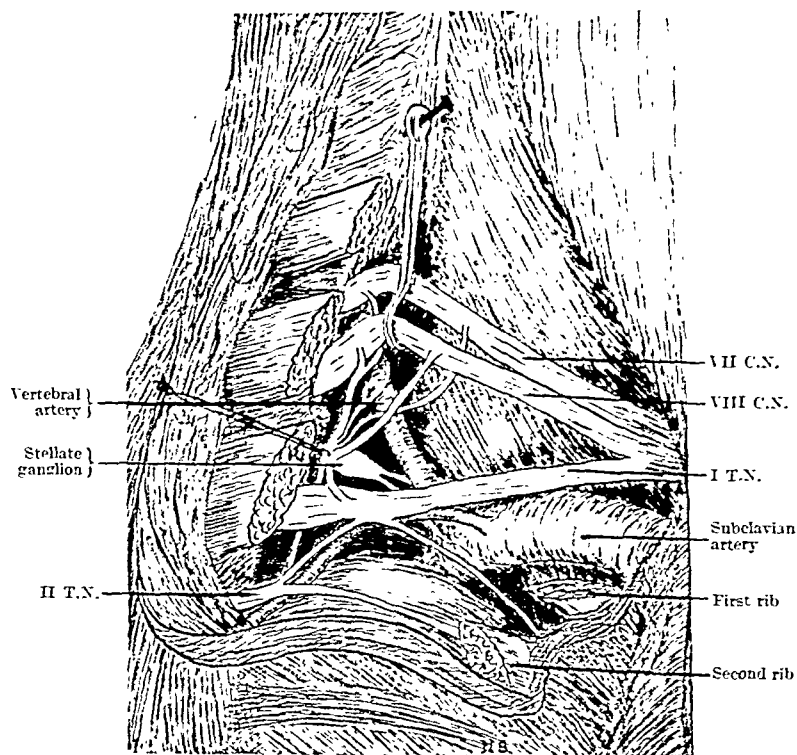
FIG. 223.—Right side. Posterior approach. Portions of first and second ribs removed. Note the position of the stellate ganglion between the eighth cervical nerve and the first thoracic nerve.

tial therefore at operation to remove the upper thoracic cord as far caudally as to include the second thoracic ganglion. By this means only can all the pre-ganglionic fibres to the upper limb be securely cut. In order to ensure this low excision, the dorsal route for the operation is essential, removing a portion of the first rib and preferably a part of the second (*Fig.* 223). This point is mentioned, as the anterior route is still practised by some surgeons in this country.

In performing this posterior approach the anatomical relationship of the sympathetic cord to two structures in particular appears important—namely,

the superior intercostal artery and the thoracic duct. The superior inter-
costal artery has been preserved in several of the dissections and is illustrated
in *Figs.* 222, 224–226. It arises above the inferior cervical ganglion from
the posterior aspect of the subclavian artery. It arches laterally and dorsally
over the upper part of the ganglion, gives off its deep cervical branch,
and then itself continues downwards along the lateral border of the inferior
cervical and first thoracic ganglia, crossing over ventrally the eighth cervical
and first thoracic nerves in this order. It frequently gives off a branch from
its medial aspect, which passes posterior to the intervening sympathetic cord
between the first and second thoracic ganglia. Not only is it important to
recognize and secure this artery, or one of its branches, at the time of operation,
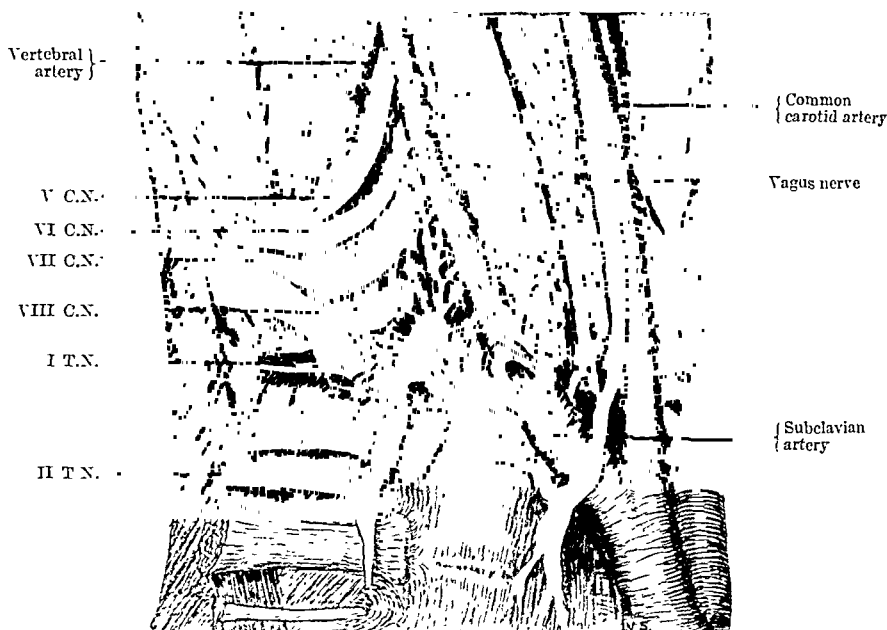
Fig. 224.—Right side. Anterior tubercles of transverse processes of sixth and seventh
cervical vertebræ removed to show the vertebral plexus. The relationship of the superior
intercostal artery can also be seen.

but it also may serve as a very helpful guide to the exact position and direc-
tion of the upper thoracic cord. The accompanying vein may be represented
as two venæ comites, but more frequently it passes down medial to the artery,
lying between the latter and the sympathetic cord (*see Fig.* 222). In this
position it is therefore in greater danger of being damaged than the artery.
    The important relationship of the inferior cervical ganglion to the
thoracic duct has been emphasized by Minkin[28] in relation to cervical sym-
pathectomy by the anterior approach. By this route the bend of the thoracic
duct must be defined and carefully avoided as it hooks round on the ventral
aspect of the stellate ganglion, from which it is separated by a thin layer of
fatty tissue traversed by numerous small veins. The arch of the thoracic

Fig. 225 labels:
Sympathetic cervical cord
V C.N.
VI C.N.
VII C.N.
VIII C.N.
I T.N.
II T.N.
III T.N.
Innominate artery
Vagus nerve and recurrent laryngeal nerve

Fig. 226 labels:
artery
Trunks of brachial plexus
Vagus nerve
II T.N.
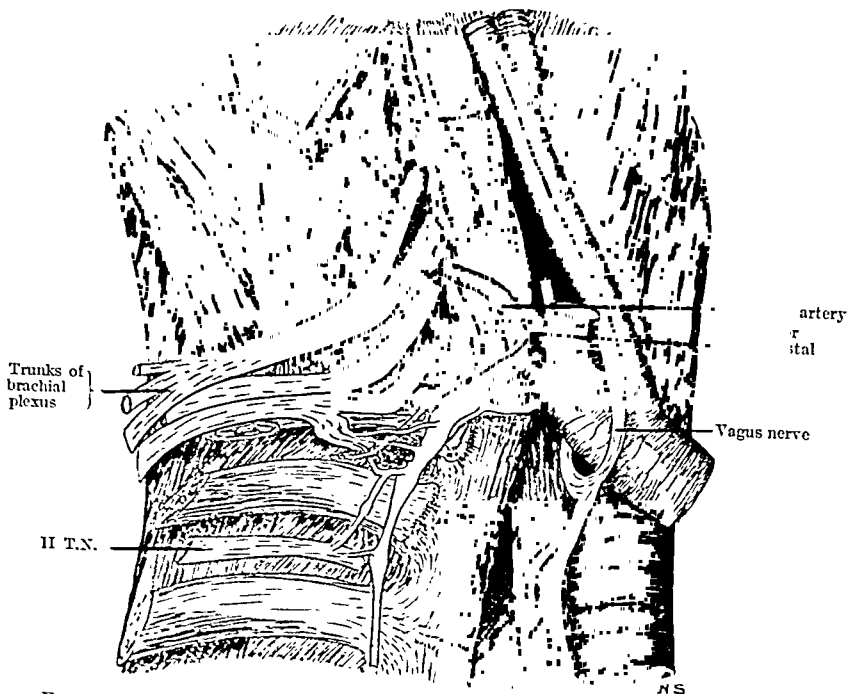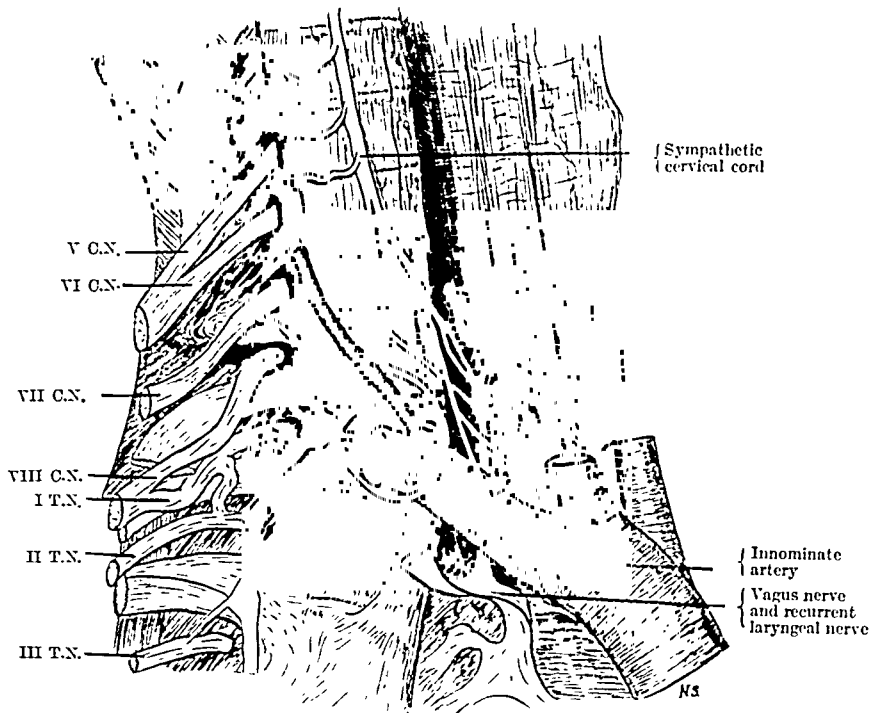
Figs. 225, 226.—Right side. Showing two typical appearances of the inferior cervical ganglion and its branches.

duct, according to Minkin, may lie higher or lower than, or completely lateral to, the stellate (or inferior cervical) ganglion. The lymphatic duct and its tributaries may closely simulate small veins, and may occasionally be ligatured and divided accidentally, under this impression. By the posterior approach for cervico-thoracic sympathectomy it would appear that the thoracic duct is very rarely in danger of being involved. The upper thoracic sympathetic cord is always reached first, the lymphatic duct, from this aspect, lying deeper. Furthermore, at this level the sympathetic cord is inclining backwards to lie directly on the ventral surface of the ribs, whilst the thoracic duct is arching forwards and therefore away from the stellate ganglion.

The contribution from the middle cervical ganglion to the brachial plexus consists of rami communicantes to the fourth and fifth cervical nerves, and in some cases it was observed to give a twig to the sixth. Occasionally these branches arose direct from the sympathetic cord, either below or immediately above the middle cervical ganglion. Much of the difference in opinion concerning the incidence of this ganglion and its exact position appears to be due to a confusion in nomenclature rather than to any particular difference in observation. The usual description is that the middle cervical ganglion, when present, lies opposite the level of the sixth cervical vertebra immediately anterior or posterior to the inferior thyroid artery, and that it is formed by the coalescence of the fifth and sixth ganglia. This is frequently referred to as the 'thyroid' ganglion. But there is a second—and from the present study it would appear a more constant—ganglion, situated on the lower cervical chain immediately ventral to the root of the vertebral artery. It is connected with the inferior cervical ganglion usually by two short stems which encircle the vertebral artery. This has been called the 'vertebral' ganglion by some, and by others the 'intermediate' ganglion (Jonnesco). From the present study one or other of these ganglia was almost invariably present, though occasionally very small in size ; and it seems better to follow the description as given by Axford and consider them both as constituting the middle cervical ganglion, and to distinguish them by the terms 'high' and 'low' types respectively. In those cases where they occur together, the middle cervical ganglion is obviously double. Laubmann divides the arrangement of the cervical sympathetic cord into five main types, and by far the commonest was that in which the superior, middle (either 'high' or 'low' type), and inferior cervical ganglia were all present and distinct. The high type of middle cervical ganglion is crossed, usually anteriorly, by the inferior thyroid artery just where the latter loops downwards to meet the recurrent laryngeal nerve. In addition to the rami communicantes described above, the ganglion gives off the middle cardiac nerve, branches along the inferior thyroid artery, and occasionally, as seen in *Fig.* 227, a communicating branch to the phrenic nerve. The ansa subclavia may come from the 'high' or 'low' types of the middle cervical ganglion or from the sympathetic cord itself.

The inferior cervical ganglion is partially or entirely fused with the first thoracic ganglion in about 80 per cent of cases (Testut), the united mass being known as the 'stellate' ganglion or ganglion of Neubauer. All degrees of this fusion can be seen (*Figs.* 224–226). A complete union would appear from the present study to occur in only about 50 per cent. An important

alteration in the direction of the sympathetic cord occurs at the inlet to the thorax. In the lower part of the neck the cord lies in a direction downwards and with a slight inclination medially. This ends at the so-called 'vertebral' or low type of middle cervical ganglion, and from here the inferior cervical ganglion turns backwards over the transverse process of the seventh cervical vertebra and over the neck of the first rib. The continuation of the upper thoracic cord is at first backwards as well as downwards and laterally. It then runs along a line drawn at right angles to the long axis of the ribs about half an inch from the costovertebral articulations.
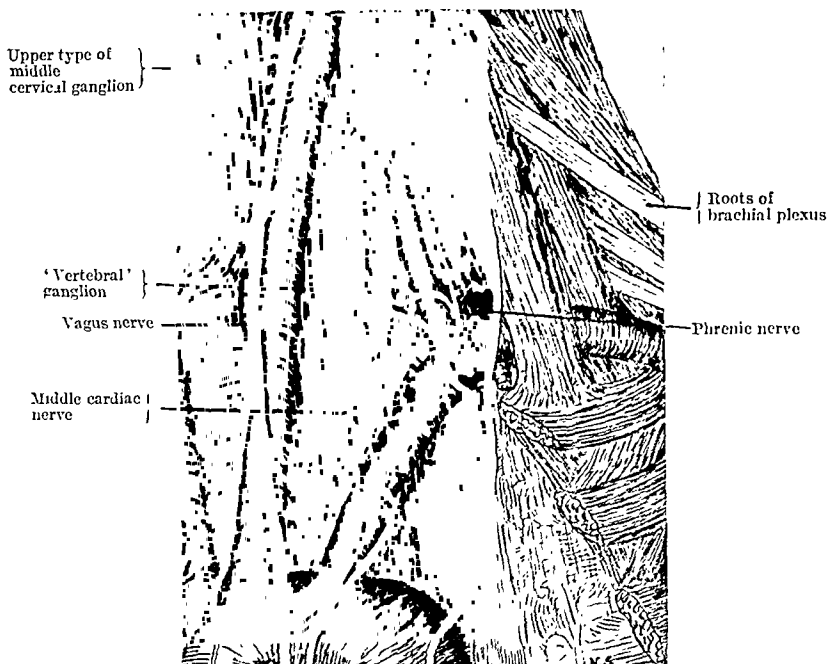
FIG. 227.—Left side. Showing the appearance before removing a portion of the sub-clavian artery. Note the presence of both 'high' and 'low' types of middle cervical ganglion. There is also a connection between the sympathetic and the phrenic nerve. This figure illustrates the extensive peri-arterial plexus around the origin of the subclavian artery.

The stellate ganglion lies in a niche posterior to the pleura and to the subclavian artery and sometimes to the origin of the vertebral artery, and anterior to the transverse process of the seventh cervical vertebra and the neck of the first rib. In the high position it may actually present a groove or depression on its anterior aspect, in which the vertebral artery rests. The stellate or inferior cervical ganglion is constantly to be found between the eighth cervical and first thoracic nerves, which it crosses on their ventral surface. In size the inferior cervical ganglion has been found never to measure less than 1 cm., and in those cases where it is united with the first thoracic ganglion its length may exceed 2 cm. A simple idea of its thickness can be obtained by remembering that it is usually at its widest part not very much

less than the eighth or first thoracic nerves which it is crossing. No useful purpose is fulfilled by attempting to assign any particular form or shape to the ganglion. It will be seen from the accompanying figures that occasionally it is truly stellate, but more often triangular or even an elongated inverted cone (*Figs.* 224-226).

The branches of distribution from the present study of the stellate ganglion can best be briefly summarized as follows :—

1. **Rami Communicantes.—**

a. Superficial or Direct Branches.—*To fifth, sixth, seventh, and eighth cervical and first thoracic nerves. These all took origin from the upper and lateral aspects of the ganglion. Often they were double or divided into two or three branches as they reached the spinal nerve. They usually passed behind the superior intercostal artery except those to the eighth cervical nerve, which frequently crossed on the ventral aspect of the artery. For a detailed account of the exact course and relationship of each of these rami, the excellent paper by Axford should be consulted. The particular significance of the grey rami to the first and second thoracic nerves has already been emphasized.

*b. Deep or Indirect Branches from the Vertebral Nerve.—*To fourth, fifth, and sixth cervical nerves.

2. **Nervus Vertebralis** arose from the superior aspect of the ganglion. It was very constant. It followed the vertebral artery on its lateral side, and with the latter entered the vertebral foramina in the cervical transverse processes. As it passed upwards it gave off important twigs to the vertebral plexus around the artery, but its largest branches were given off to the sixth, fifth, and often fourth cervical nerves as they crossed behind the vertebral artery. *Fig.* 224, in which the anterior tubercles of the transverse processes are shown removed, demonstrates this very clearly. These 'deep' rami communicantes would of course degenerate in a cervico-thoracic ganglionectomy, but the close association of the nerve with the vertebral artery would make it extremely difficult to ensure its division in a ramisection.

3. **Ansa Subclavia** (*Ansa Vieussens*)† arose above from the middle cervical ganglion or the lower cervical sympathetic cord, crossed over the first part of the subclavian artery, and wound round the lower aspect of this in a long course to reach the anterior surface of the inferior cervical ganglion. Because of its attachment to the ventral aspect of the ganglion, and on account of its very close relationship to the subclavian artery, it is in great likelihood of being missed in any operation of ramisection by the dorsal route. It was frequently double, and may form a connection with the phrenic and with the recurrent laryngeal nerves (*see Fig.* 227). This has been emphasized by Shawe,[31] who found it present in 68 per cent of the specimens investigated. It distributes branches to the peri-arterial nerve plexus around the subclavian artery.

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* Following the method of description as given by Hovelacque.

† Hovelacque has pointed out that Vieussens (1684) actually described a communicating branch uniting the inferior cervical and the first thoracic ganglia, in front of the subclavian artery.

4. **Direct Vascular Twigs** to the subclavian artery and its branches, particularly the vertebral.

5. **Short Stout Communicating Branches** surrounding the origin of the vertebral artery, and uniting on its ventral aspect in the 'vertebral' or 'low' type of middle cervical ganglion or in the cervical cord itself.

6. **Inferior Cardiac Nerve** arose from the supero-medial aspect of the inferior cervical ganglion. or frequently in common with the ansa subclavia. In the present study it passed most commonly, both on the right and the left side, along the lower and posterior aspect of the subclavian artery, to the deep cardiac plexus. It nearly always formed a plexus of communication. and in some cases actually blended with the middle cardiac nerve and the recurrent laryngeal nerve. This plexus surrounds the origin of the sub-clavian artery, particularly posteriorly, to which it gives numerous branches (*see Fig.* 227). This is only illustrated by Hovelacque (*Plate XCIX. p.* 676) and by Jonnesco (*Fig.* 1. p. 92). In this connection Jonnesco gives the follow-ing very significant passage : " Dans leur long trajet, les nerfs cardiaques s'anastomosent entre eux, avec les rameaux cardiaques du pneumogastrique, et avec les rameaux du laryngé supérieur et inférieur. Quelquefois, ils s'approchent l'un de l'autre en changeant de fibres, directement, et forment un plexus. D'autres fois, au niveau de leur contact on trouve un gan-glion." The cardiac nerves often present along their course one or more ganglia. According to Jonnesco three are more or less constant. one at the point where the superior cardiac nerve anastomoses with the middle, a second along the course of the superior cardiac nerve, and a third on the course of the middle cardiac nerve in its thoracic portion. It is an important and as yet unanswered question how far this intimate plexus between the sympathetic cardiac nerves and the vagus (and occasionally the phrenic) enters into the innervation of the blood-vessels of the upper extremity. Nor can the presence of these small ganglia in this plexus which surrounds the first part of the sub-clavian artery be entirely overlooked. There appears here to be a possible difference in the anatomical arrangement of the vascular nerves in the upper and lower extremities.

In conclusion it would appear that if one could be certain that the peri-vascular plexus around the subclavian artery is entirely sympathetic, and if one can exclude the possibility of vascular reflexes through remaining sympathetic ganglia. then it should be possible to denervate completely the sympathetic supply of the upper limb by removing the stellate ganglion and the upper thoracic cord as far down as to include the second thoracic ganglion. By this means all the pre-ganglionic fibres should be severed.

SUMMARY.

1. The difference in the upper and lower extremities in the operative results of sympathectomy for vascular lesions has been emphasized.

2. The various theories that have been advanced to explain this differ-ence have been reviewed.

3. The anatomical arrangement of the proximal source of vascular nerves to the upper extremity has been investigated on twenty-five sides.

4. Certain important surgical and anatomical features in relation to the operation of cervico-dorsal sympathectomy are recorded.

5. The dorsal route is the only possible method of ensuring complete sympathetic denervation of the upper limb, and the operation of ganglionectomy would appear to be a much surer and more practical method than that of ramisection.

My thanks are due to Professor J. S. B. Stopford for allowing me to carry out this work in his department, and above all for his kind help and advice. For permission to examine their clinical cases, both before and after operation, I am indebted to Professor Telford and Professor Stopford. I also wish to express my most sincere gratitude to Mr. N. Shtetinin, third-year medical student, for the excellent illustrations which he has so kindly made from my dissections.

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A REPORT ON THE STRANGEWAYS COLLECTION OF RHEUMATOID JOINTS IN THE MUSEUM OF THE ROYAL COLLEGE OF SURGEONS.

By R. LAWFORD KNAGGS,

With Photomicrographs by G. H. RODMAN.

PART III.

THE PATHOGENESIS OF GOUT.

I. THE ARTHRITIC PHENOMENA ASSOCIATED WITH GOUT.

In Part I attention was drawn to the similarity of many of the signs present in gouty joints to those which distinguish osteo- and rheumatoid arthritis, and to the impossibility of distinguishing specimens of the latter disorders from those of gout, without the help of a urate of soda deposit. At the same time the question of the toxic origin of these arthritic changes was also considered.

Now, in joints that have been affected by gout for a number of years there are usually found 'mixed' arthritic changes, such as lipping, grooving. eburnation, and bony and fibrous ankylosis, with microscopic evidence of fibrous degeneration of bone and cartilage. This association of hypertrophic and degenerative signs is present in almost every one of the Strangeways specimens of gouty joints, and is probably to be explained by the steady diminution of the vitality of the joint tissues as the disease progresses.

Gout is usually met with in fairly healthy people, and its onset, as a rule, occurs in the middle period of life, before the bodily powers have shown a tendency to wane. Therefore, apart from the inflammation set up by the crystalline deposit, the arthritic signs may be expected to take on the osteo-arthritic form. Indeed, nearly every specimen of gout in the Collection presents hypertrophic bony phenomena. These overgrowths. though they may undergo internal changes, are practically permanent. As health declines, and the joint becomes further damaged owing to the increase of the gouty deposit and the associated inflammation. degenerative changes set in. These are of the same nature as those met with in rheumatoid arthritis, and are characterized by fibrocellular degeneration of articular bone and cartilage, in which the hypertrophic formations are not only involved, but are, often, the first to suffer.

The inflammation set up by the gouty deposit, however, introduces an element of confusion. It is probably present during the evolution of the hypertrophic formations. and may account for the occasional development of bony ankylosis in the large joints, as well as in the small ones of the hands
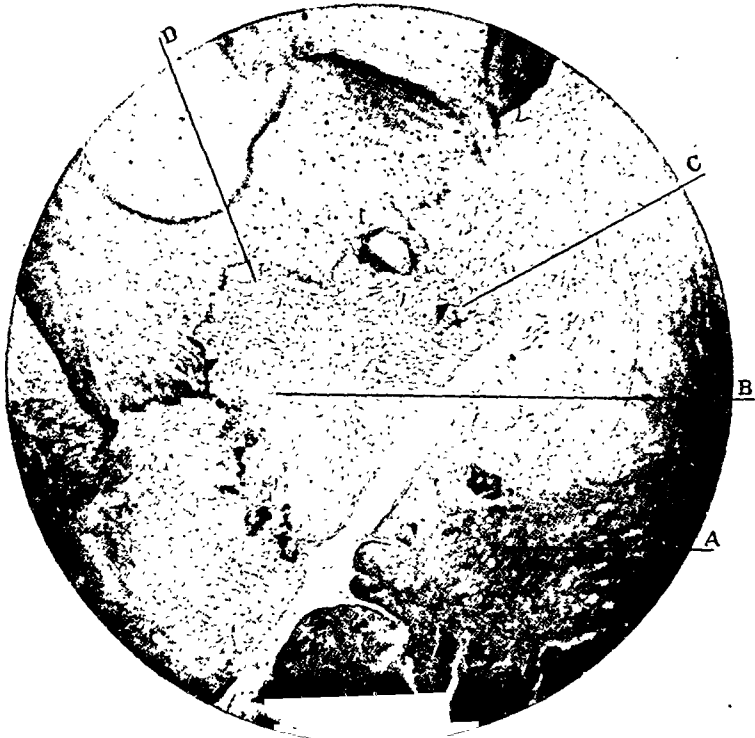
Fig. 228.—Charcot's disease. From an elbow; section through the capitellum showing fibrous degeneration of bone *en masse*. A, Degenerating and disintegrating articular cartilage; B, Massive degeneration of sclerosed bone into a very delicate network of fibrous tissue, permeated by very small spaces—probably cavities from which bone cells have disappeared, while others—larger and in groups—suggest early fat formation; C, Remains of osseous débris; D, Irregular bone edge showing continuity in many places with the fibrous tissue. (*Specimen* S.C. 90.) (× 75.)
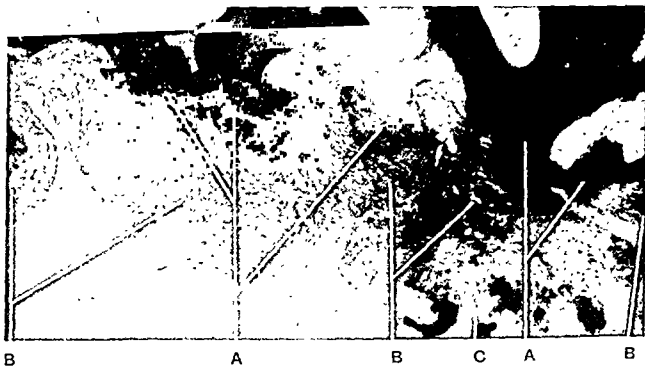
Fig. 229.—Charcot's disease. Section from articular surface of the humerus—from the same elbow as *Figs.* 228 and 234. A, Degenerating superficial portions of sclerosed bone; B, Fibrous covering, evidently largely produced by fibrous degeneration of bone; C, Fibrous villi. (*Specimen* S.C. 90.) (× 50.)

and feet, without invoking the peculiar activity of the cartilage cells, which precedes bony ankylosis in rheumatoid arthritis.   It may also be expected to intensify the inflammatory changes usually associated with rheumatoid arthritis, and by its damaging influence on the joint tissues to predispose to the onset of degeneration.

It is not surprising that inflammation in gout is more acute and widespread than in rheumatoid arthritis, for urate of soda is deposited far beyond the articular surfaces.   It may be found in the capsule, the bursæ, the tendons, the periosteum, and even in the trabeculæ ; and deposits may occur in the medulla well outside the limits usually involved in rheumatoid arthritis.   In the latter situation they may be of considerable size.   Degeneration, and absorption of bone by granulation tissue, may even lead to the formation of cavities in the cancellous tissue, and these may be packed with urate crystals (S.C. 74).

In nearly every instance the gouty deposit is probably the first pathological lesion to develop.   Yet this is not always the case, and the Collection contains one example from an old woman aged 85 who had been a typical case of rheumatoid arthritis for many years—certainly twenty, and probably more ;   both knee-joints show pronounced evidence of that condition, but in the left there are a few patches of urate of soda in places (S.C. 83).

II. THE MICROSCOPICAL CHARACTERS OF THE GOUTY DEPOSIT.

Urate of soda crystals seem to give rise to little or no irritation when in small collections in the cartilage ;   but in the capsule, synovial membrane, and the bone medulla, they do.   Inflammatory cells are often present in the neighbourhood of deposits in the synovial membrane and capsule, and uratic masses are frequently found in the medulla in the centre of an area of granulation tissue. Within such an inflammatory focus trabeculæ, or portions of trabeculæ, are sometimes undergoing fibrous resolution.

In synovial villi small deposits may have a circle of elongated connective-tissue cells around them, suggesting an attempt at encapsulation.   (*Fig.* 230.)

Giant cells are not infrequently present in the immediate neighbourhood of deposits, and often several may be closely applied to one.   An interesting appearance was seen in connection with the remains of the articular cartilages of an astragalo - calcanean joint from a

FIG. 230.—Gouty deposits of urate of soda in synovial villi. (*Specimen* S.C. 65.) (× 50.)

woman aged 76, in whom both bones were fused.   The articular cartilages lay in the midst of fat medulla occupying the spaces of a very atrophic cancellous tissue ;   they were undergoing fibrocellular degeneration with the production

FIG. 231.—Crystals of urate of soda in the capsule of an elbow-joint. They have the usual yellow-brown colour characteristic of urates, more particularly at the thicker parts of the rosettes.
(*Specimen* S.C. 67.) (× 85.)

FIG. 232.—Thick deposit of urate of soda crystals in, and on the inner surface of, the capsule of an elbow joint. Owing to the thickness of the deposit, it presents a dark brown colour. Under a high-power its component crystals are easily seen.
(*Specimen* S.C. 67.) (× 50.)
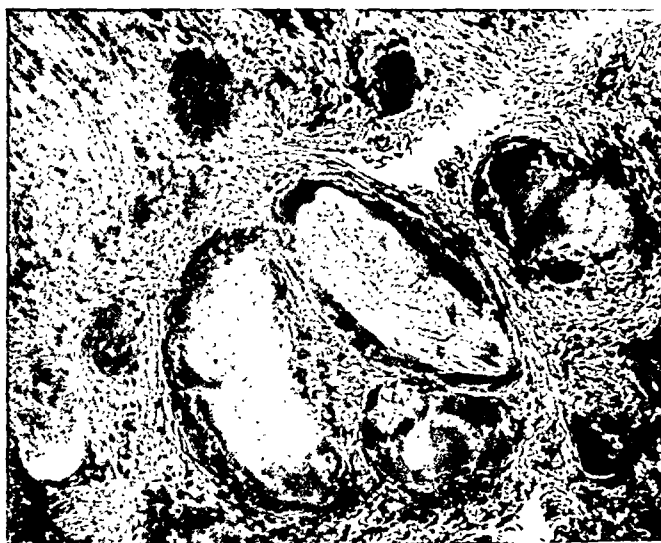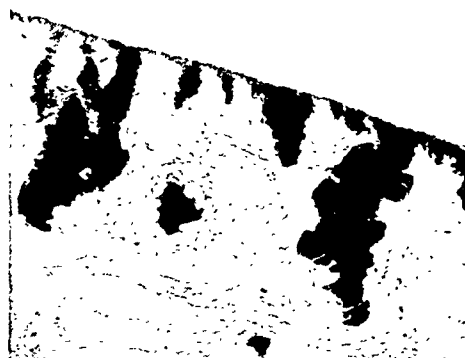
FIG. 233.—Deposits of urate of soda in an inflammatory mass which has penetrated a patella from its dorsal surface and appears to have isolated a portion of the bone. The deposits tend to become encapsulated and have a number of giant cells on their edges. The crystalline texture of one of the large masses is seen in the photograph. From a woman, aged 76. (*Specimen* S.C. 25—2.) (× 100.)

of large numbers of giant cells; some urate of soda deposits of good size, which had possibly been liberated by the dissolution of the cartilage, were surrounded by giant cells, which in one instance were so numerous that the mass was encircled with a row of cells like a necklace of beads (S.C. 81—1). Under the microscope, urate of soda deposits may appear as well-defined, structureless, white or grey patches. In the superficial layers of the cartilage these may be very small, and sometimes small chains of granules may be seen near them in the same layers—possibly these granules are not crystalline. Larger deposits form white masses having a somewhat hazy and granular look. On careful focusing with a high power a delicate fibrillar network may be detected in them in some instances. The needle-like crystals of which the deposits are composed can sometimes be distinguished. but as a rule they cannot, probably because the decalcifying process has caused some change in their composition, or has dissolved them out to some extent. In some instances the crystals may show the yellow-brown colour typical of urates. This is more likely when the deposit is copious and thick. Sometimes beautiful star-shaped masses of crystals may be seen. in which the colour is most pronounced at the nodal or thickest parts. whilst the rays show little of it. In others, a long tract of dark-stained material may infiltrate the surface layers of some joint structure such as capsule or articular cartilage, and its crystalline nature may be revealed by a high power.

THE PATHOGENESIS OF CHARCOT'S DISEASE.

I. THE HISTOLOGY OF CHARCOT'S JOINTS.

When the literature of Charcot's joint disease was investigated a few years ago very little information was found about the histology. Mott—apropos of fractures in tabes—had shown that the Haversian canals were irregularly dilated and filled with fat (*System of Syphilis*, iv. 366-370), and Targett had pointed out that the ragged-looking membrane, presenting numerous villous processes and polypoidal masses, which lined the interior of the joint cavity, and was more or less continuous over the eroded bone-ends, was of fibrocartilaginous structure, and that the polypoid bodies were formed by cartilage, in part hyaline, and in part fibrous, whilst calcification was going on in the centre (*Pathol. Soc. Trans.,* xlii, 270). Neither of these observations dealt with the microscopical changes in the articular ends of the bones, and they leave untouched the vague impression that the erosion of the bone-ends is due to mechanical attrition of parts from which trophic influence has been cut off.

Nine cases of Charcot's disease are represented in the Strangeways Collection. and there are numerous microscopical sections obtained from as many as thirteen joints. The study of these revealed a number of interesting facts.

A description of the histology of Charcot's joints would, for the most part, be simply a repetition of the account of the degenerative changes in rheumatoid arthritis. The articular cartilage, in a very early stage of its degeneration. shows a villous surface, numerous foci of aggregated cells, and
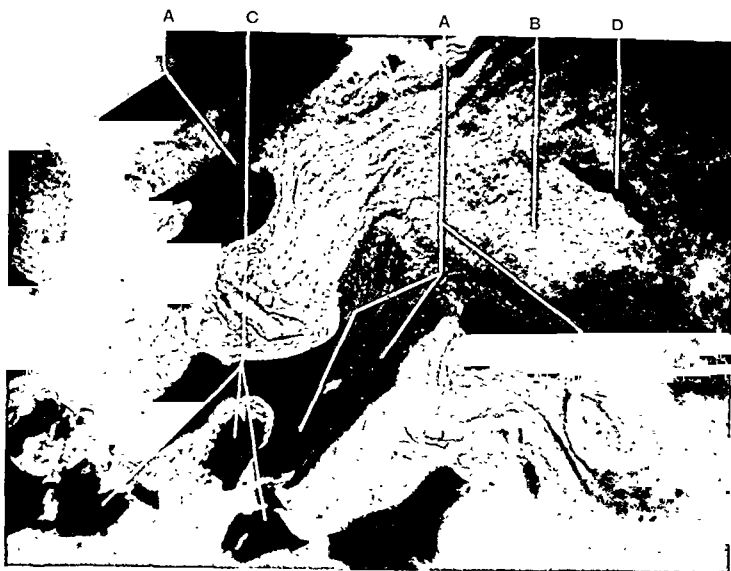
FIG. 234.—Charcot's disease. Section of the articular surface of an ulna—from an elbow. No history. A, Degenerating trabeculæ ; B, Fibrous covering formed from fibrous degeneration of bone ; C, ? Artefacts, which would be easily caused ; D, Portion of a trabecula in the fibrous covering in process of resolution. (*Specimen* S.C. 90.) (× 50.)
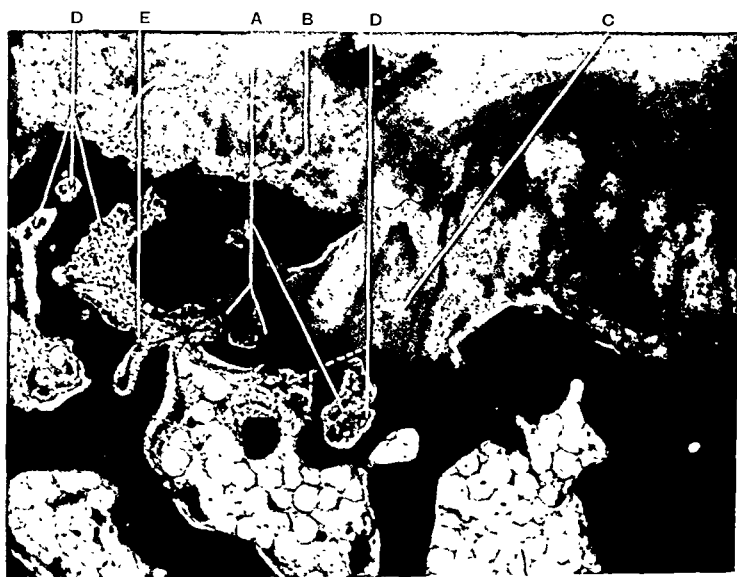
FIG. 235.—Charcot's disease—an early condition in a knee. The photograph shows the only part in a section through an external femoral condyle where characteristic changes occurred. The section included a small quiescent marginal osteophyte containing fat marrow. A, Degenerative spaces in bone ; B, Atrophic cartilage ; C, Degenerating and atrophying cartilage merging in indistinct bone ; D, Giant cells ; E, Compound space communicating with the marrow. (*Specimen* S.C. 93—2.) (× 50.)

spaces in process of formation. It is easy to understand its speedy disappearance, and often the only trace of it may be a mere strip of fibrous tissue.

FIG. 236.—Charcot's hip. Section from an eroded femur showing a curious appearance presented by the atrophying bone. V indicates the part which is enlarged in *Fig.* 237. (*Specimen* S.C. 86.) (× 6.)

In the articular ends of the bones the essential change is fibrous degeneration. There is the same formation of spaces in the bone and in the cartilage, the same resolution of those structures into fibrocellular tissue, and the same development of giant cells, as in rheumatoid arthritis. But there is this difference in Charcot's disease—the degenerative changes are more pronounced and more widely spread. Not only are large blocks (microscopical) of fibrous tissue seen in places that have previously been occupied by bone or cartilage, but the evidence of degeneration and resolution extends to much greater depths. Indeed, degeneration sometimes seems to take place *en masse*.

There can be no doubt that this degeneration of cartilage and bone is responsible for the rapid erosion of the articular ends of the bones, which is one of the most striking features of Charcot's disease.

An interesting variation in the form of fibrous degeneration of bone was first noticed in a section from a Charcot's

FIG. 237.—Charcot's hip. The illustration shows a portion of *Fig.* 236 (V). The degenerating trabeculæ form a network of anastomosing channels owing to the disappearance of their central parts. The walls of the channels show traces of lime (hæmatoxylin stain), and at A a persisting portion of bone projects into the lumen where three channels join. (× 40.)

hip ; a network of trabeculæ had been converted into a series of anastomosing channels owing to the degeneration, and disappearance, except for some fine fibrils, of their central portions. The walls of the channels were formed of fibrous tissue along which traces of calcareous matter, and occasionally small portions of bone, appeared. Anastomosing fibrous strands in the fat medulla were probably relics of vanished trabeculæ. This peculiar type of fibrous degeneration of the bone trabeculæ was also observed in several cases of rheumatoid arthritis (S.C. 23—1 ; S.C. 27—1 ; S.C. 32 ; S.C. 35—14).

It has long been recognized that the extensive destruction which takes place in a Charcot's joint is induced by the withdrawal of a trophic function. This is an acknowledgement of the part that vitality of the articular tissues
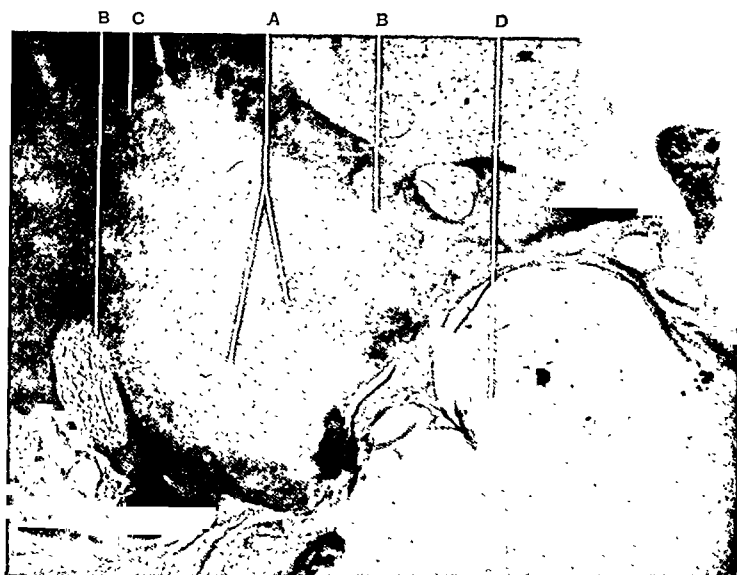
FIG. 238.—Charcot's disease. Section from the articular surface of the head of the humerus—from the right shoulder of Philip H., age 50. A, Degenerating areas in a sclerosed trabecula ; B, Spaces which are in process of extension by continued degeneration of portions of their margins : C, Sclerosed bone, which (outside the picture) is covered by a considerable thickness of fibrous tissue resulting from bone resolution ; D, Cystic space, whose bony margins are recruiting the fibrous tissue which lines it. (*Specimen* S.C. 89—2.) (× 50.)

plays in this affection. The similarity of the microscopical changes suggests that the only etiological difference between rheumatoid arthritis and Charcot's disease is, that in the latter the innervation of the joint has been impaired. The much greater destruction in the case of a Charcot's joint may be explained by the more extensive degeneration, and by the absence of pain permitting free movements.

It might be argued that Charcot's joint disease is simply rheumatoid arthritis in a patient who suffers from tabes ; but the nerve defect, associated with the more widely spread degeneration, and consequent inordinate destruction, produces a combination of symptoms so unique that it would be absurd to think of grouping the two conditions together. It is sufficient if the similarity of the microscopical changes is recognized and the relationship admitted.

II. OSTEOPHYTES AND CAPSULAR OSTEOPHYTES IN CHARCOT'S DISEASE.

It was the remarkable development of osteophytes that caused the early controversy as to the nature of Charcot's joints, and furnished the best argument for those who regarded the affection as osteo-arthritis in a tabetic. But the formation of osteophytes seems incompatible with the rapid degeneration of the articular bone and cartilage that takes place in these cases. There are, however, two kinds of osteophytes—those growing in connection with
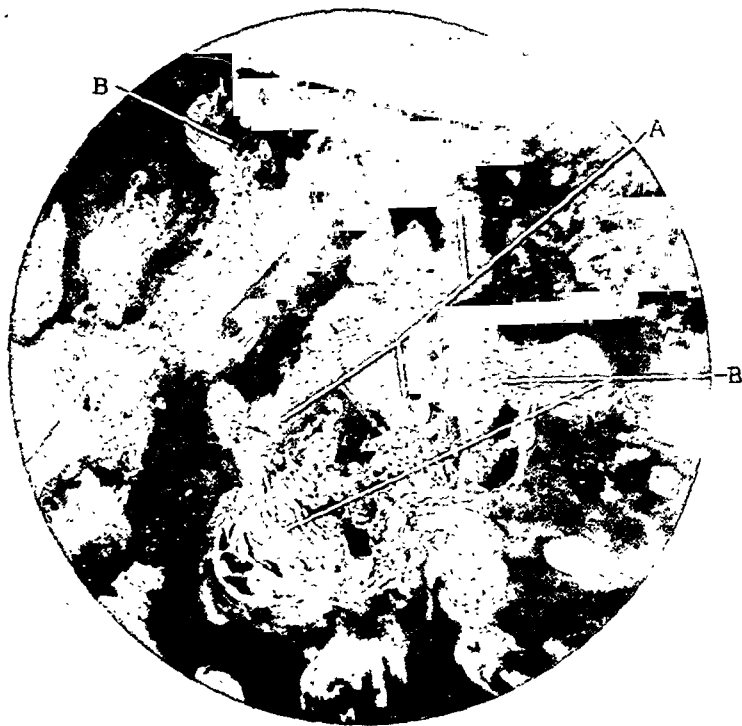
Fig. 239.—Charcot's disease. Section from a knee—not in the Collection, but described particularly in the Strangeways MS., *Fig. 104A.* It shows widespread degeneration of the articular surface. A, Massive degeneration of bone in a sclerosed and eburnated area, fibrous tissue becoming evident : B, Advanced stage. Spaces containing coarse fibrous tissue, resolving fragments of bone, and small quantities of small fat globules. (*Specimen* S.C. 85.) (× 40.)

the articular cartilage, and those forming in the capsule. The latter are numerous in Charcot's disease, and as they have a different mode of origin from articular or marginal osteophytes, the incompatibility just mentioned may be capable of explanation.

The result of a special examination of the Charcot's joints in the Strangeways Collection, and in the Hunterian Museum, showed that though there was no clear evidence of the presence of osteophytes of the articular surface or margin, yet the extravagance of the new bony formations made it impossible to be sure that they did not exist.

FIG. 240.—Charcot's disease. From the same joint as *Fig.* 239. Degenerative changes in sclerosed and eburnated bone. Note the spaces with jagged margins (A). They contain many fragments of degenerating trabeculæ which tend to end in débris and fibrous tissue. Cells are scanty or absent. There are no giant cells. The presence of vessels is doubtful. (× 40.)

FIG. 241.—Massive fibrous degeneration of bone in Charcot's disease. Section from a femur—Charcot's knee. A, Sclerosed trabecula showing several areas of central degeneration. B, A mass of fibro-connective tissue containing, near the surface, relics of trabeculæ undergoing fibrous resolution — also connective-tissue cells, vessels, and inflammatory cells. It extends deeply between two degenerating sclerosed trabeculæ, and is covered by a thick layer of dense fibrous tissue, containing many connective-tissue cells and some vessels.

(*Specimen* S.C. 88—1.) (× 50.)

Several microscopical sections, however, show osteophytes. Three are almost certainly capsular osteophytes, which have fused with the articular margin (S.C. 85; S.C. 88—1; S.C. 89—1). Another is of doubtful nature. and the fifth—a small marginal osteophyte in an early case—has no doubt developed in the usual way. It shows no sign of recent growth. the medullary spaces are of good size and regular, the trabeculæ are well formed. and the marrow is fatty; but early signs of degeneration have made their appearance in the joint. and the bony surface of the osteophyte shows definite evidence of fibrous degeneration (S.C. 93—2). Evidently the degenerative process has attacked an osteophyte that had settled down to permanency before Charcot's disease developed in the joint. These instances may be contrasted with another specimen (S.C. 22). which is a pretty example of marginal lipping of femur, tibia. and patella with epiarticular osteophytes of the femoral condyles. The patient was a woman, aged 63, who had had rheumatic fever at the ages of 12 and 24, and had suffered from tabes for many years. The knee was swollen and contained fluid and was regarded as a 'typical Charcot's knee'. The left leg had been amputated ten years before for a useless swollen knee, also diagnosed as a Charcot's joint. The specimen, however. has been catalogued as 'osteo-arthritis in a tabetic'. for there is no sign pointing to Charcot's disease, either macro- or microscopically.

These facts are opposed to the idea that articular osteophytes are common in Charcot's joints; and it is difficult to avoid the conclusion that the osteophytes usually met with in such cases are capsular formations. Capsular osteophytes, when they are situated near an articular margin. may become attached to the bones; in some cases owing to their increase in size, and in others by ossification in the connecting fibrous tissue of the capsule.

Marginal osteophytes of the ordinary type, met with in definite neuro-pathic joints, are almost certainly of pre-Charcot development. They will probably show degenerative changes resulting from the tabetic affection.

The origin of capsular osteophytes is of more than ordinary interest, but to avoid repetition it is considered with sclerosis and eburnation, to which it is closely related.

III. SCLEROSIS AND EBURNATION IN CHARCOT'S JOINTS.

Some sclerosis of the joint ends of the bones is not uncommon in advanced Charcot's disease, but eburnation is rare. The Strangeways Collection contains two remarkable macerated knee-joints, in which extensive polished ivory-like surfaces are present on several bones (S.C. 88; S.C. 92). It can hardly be conceived that bone which is the subject of degeneration and fibrous resolution can undergo sclerosis and eburnation any more than that it can develop osteophytes; but there is no doubt that the articular ends of the bones entering into a Charcot's joint, in which destruction has been extensive, may present eburnated surfaces; and that the dense bone may be of considerable thickness. How is this to be explained?

When cartilage or bone is the subject of fibrous degeneration, it is the matrix that is destroyed; and the fibrous tissue that replaces it is derived

from the cells present in the disappearing tissues. The cells are more viable than the matrix. In most cases they proliferate and give either a very cellular or a sparsely cellular character to the fibrous tissue. In some instances the degenerative spaces, and also some of the adjacent medullary spaces, may be so crowded with cells that the fibrous tissue supporting them

Fig. 242.—Articular surface of a femur from a Charcot's knee, showing dense eburnation.
(*Specimen* S.C. 88.)

is difficult to detect; in others, cells may be numerous only along the edges of the bone. In the latter case, presumably, proliferation is not so active, or the cells tend to atrophy and disappear, owing to their feeble vitality. In still other cases atrophy overtakes the cells in their cartilaginous capsules or bone cavities, which then appear as empty spaces, whilst the fibrous tissue may be noticeable for its acellular character.
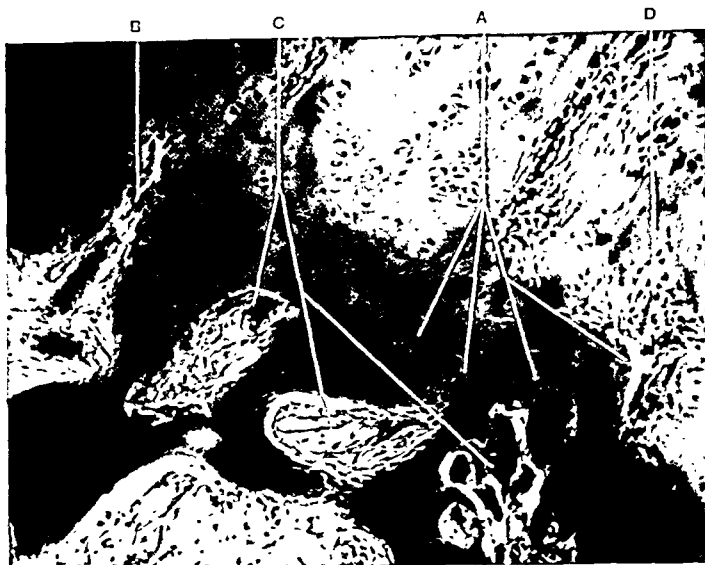
FIG. 243.—Charcot's elbow (right). Section from the head of the radius of Emily K. A. Bone degeneration in subchondroid bone layer—preparatory to formation of spaces; B, The same as A, but fibrous tissue is shown well; C, Formed spaces, two showing giant cells; D, Space forming at the expense of the calcified cartilage and bone. (*Specimen* S.C. 91—1.) (× 110.)
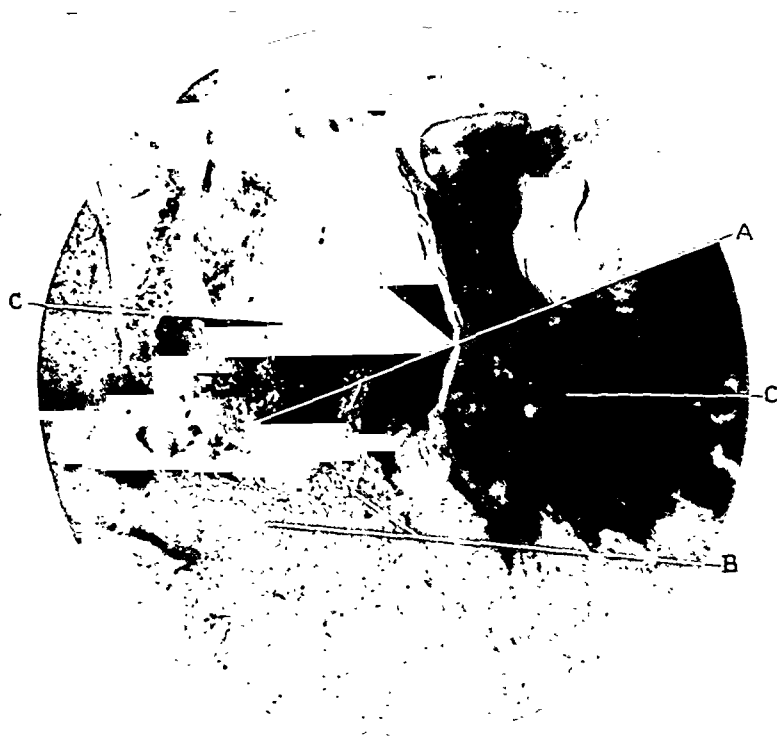
FIG. 244.—Charcot's disease. Section showing degeneration of sclerosed bone from the same knee as *Fig.* 239. A, Spaces full of fibrous tissue showing spaces containing shrivelled cells or their débris; B, Part which suggests fibrocartilage, but the cells are degenerate and the spaces enlarged and in most cases without definite contents; C, Early stage of bone degeneration. (*Specimen* S.C. 85.) (× 75.)

The causes of the degenerative changes in the articular bone-ends in Charcot's joints, are, in all probability, the same as in rheumatoid arthritis, and, as in that affection, the changes may be accompanied by more or less evidence of inflammation or even by necrosis.

FIG. 245.—Charcot's knee (left). The section is from the capsule and shows a nodule of hyaline cartilage in an area crowded with connective-tissue cells and containing many vessels. Six giant cells are applied to the margin of the cartilage, and several of the cartilage cells are surrounded by a ring of calcification. From an elderly man. (*Specimen* C.S. 88—2.) (× 50.)

The cells resulting from fibrous degeneration of bone and cartilage no doubt possess different degrees of vitality in different cases, but their subsequent behaviour may be largely determined by the kind of cell from which they have originated. Some have developed from the cells of the connective-tissue basis of the cartilage or bone, and others have been evolved from actual cartilage and bone cells. Both may be expected to inherit ancestral proclivities.

Many of these cells must be set free by the rapid disintegration of the articular surfaces and find their way into the joint. From the joint cavity they will be carried by the effusion into the synovial and capsular lymphatics, and into those of the structures in the immediate vicinity.

In either of these situations they may be held up (*Toxic and Inflammatory Diseases of Bone*, p. 112), and under suitable conditions may proliferate, and originate new ossific or chondro-ossific formations (capsular and extra-capsular osteophytes), as well as fibrous tissue. It is fair to suppose that the hypertrophic form of the disorder, which is accompanied by numerous capsular osteophytes, is associated with the cases in which the degenerative process is accompanied by a free proliferation of cells, and the atrophic form with those in which the cells disappear in an early stage of the process (*R.C.S. Museum Specimen* 4572—1).

The explanation of sclerosis and eburnation of the articular surfaces follows the same lines as that of the formation of capsular

FIG. 246.—Charcot's knee (left). Section through a bony nodule in the capsule from the same knee as *Fig. 245*. The nodule contains cancellous spaces and is growing peripherally by metaplasia of the fibrous tissue in which it lies. (× 50.)

osteophytes. From the nature of the pathological change in the cartilage and bone, it follows that the eroded

articular ends of the bones tend to become covered by an increasing layer of fibrous tissue, in which many of the cells freed from the structures which it replaces, are entangled. The fibrous covering may be excoriated in places

FIG. 247.—Charcot's elbow (left). Section from a deformed and deflected internal condyle. From Emily K., age 62. A general view of the breaking-up articular bony surface; many giant cells. The fibrous covering is composed of dense fine fibrous tissue with many connective - tissue cells, especially in the vicinity of the bone. A, Degenerating area.

(*Specimen* S.C. 91.) ($\times$ 50.)

by the rubbing of the bones, but gradually it becomes consolidated, and more or less permanent. The two cases of marked eburnation which have been mentioned show, however, that it is possible for bone to develop in this layer,
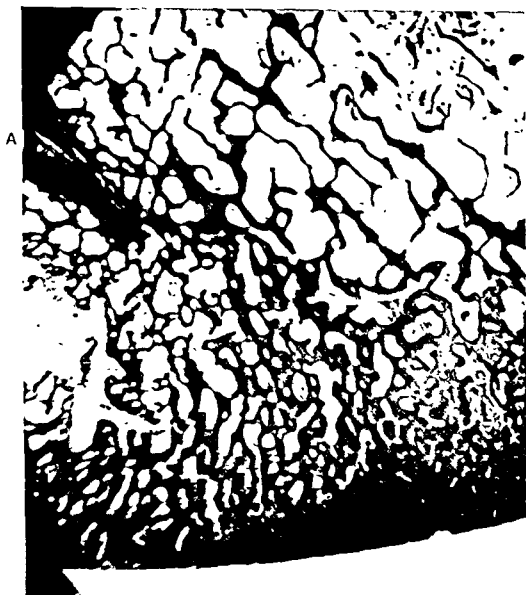
FIG. 248.—Sclerosis and eburnation in Charcot's disease (*see Fig.* 250). The section—from a femur of an old man—shows the gradual transition from atrophic cancellous tissue to marked sclerosis and eburnation, with degenerative changes supervening on the surface. The condition bears a considerable resemblance to the similar process in osteo-arthritis. Nowhere is the widespread degeneration of bone, so characteristic of Charcot's disease, to be seen. The sclerosis is advancing by metaplasia of the fibrocellular tissue which occupies the medullary spaces. Where the surface structure is degenerating there are many giant cells among the fibrocellular results, applied along the edges of the disintegrating bone. There are numerous inflammatory cells throughout the section. A, Dense bone, pointing to where a capsular osteophyte has merged with the new articular surface.

(*Specimen* S.C. 88—1.) ($\times$ 6.)

and even to go on to sclerosis and the production of a new bony articular surface. A section through the latter in S.C. 88—1 shows that there is a gradual transition from atrophic but otherwise normal cancellous tissue to

dense sclerosis and eburnation, the medullary spaces gradually becoming smaller, and being filled by granulation tissue in which ossification advances by metaplasia.

It is not easy to understand why degeneration should give place to constructive development. In the case of the *sclerosed articular surface* it may be suggested that the fibrous tissue, which replaces the cartilage and bone, is practically scar tissue, and consequently is not supplied with nerves. In an environment no longer dependent on innervation for its stability, the cells find sufficient nourishment to maintain life, and to enable them to develop according to their inherited capabilities. Eventually this results in bone formation, which may go on to sclerosis. The practically normal state of the bone below the sclerosed surface is probably explained by the protection afforded by the new, thick, osseous articular surface. This would save the underlying old bone from the constant friction and slight traumatisms which would be one of the factors tending to promote degeneration.

A similar explanation may be advanced for *capsular osteophytes*. The thick capsule of a Charcot's joint is composed largely of fibrous tissue of inflammatory origin, which has replaced the atrophied original capsule. This, again, is of the nature of scar tissue, and the emancipated cells, brought to rest in it, find a soil in which they can develop their hereditary tendencies. The extravagance and irregularity of the ossification associated with the formation of capsular osteophytes is possibly connected with the loss of the trophic function.

The *absence or rarity of osteophytes in cases of rheumatoid arthritis* which have not passed through a preliminary hypertrophic (osteo-arthritic) stage raises a point of some interest. The Strangeways Collection affords microscopical evidence that small ossific nodules may be present in the chronically inflamed capsule in that affection, but they are rare and inconspicuous.

On the other hand, in osteo-arthritis capsular and synovial osteophytes are a definite feature, and articular surfaces may in some instances be enlarged by fusion of such osteophytes with marginal ones; but they are neither so numerous nor so widespread as in Charcot's disease. The origin of the capsular osteophyte is probably the same in all three conditions, but why are they numerous in osteo-arthritis, practically absent in rheumatoid arthritis, and extravagantly large and widely distributed in Charcot's disease?

Probably several factors are concerned. Free movement of an affected joint is undoubtedly one of the most important. In osteo-arthritis there is slow destruction of cartilage and slower destruction of bone, beginning on the surface. There is considerable effusion into the joint in many cases, into which liberated cells are discharged; and the joint movements are fairly free, even though painful, until checked by muscular spasm or osteophytic locking. In rheumatoid arthritis the degenerative changes begin, and are specially marked, at the junction of the cartilage with the bone; considerable destruction may be limited to this region for some time, and the passage of cells into the joint cavity, at any rate in any considerable quantity, may be prevented by a covering of articular cartilage, which, in most instances, is not quite normal in structure. The amount of effusion is usually moderate and often negligible. The capsule and ligaments are frequently thickened or atrophic and as a rule contracted. The joint movements are painful from

the start, soon become restricted, and finally abolished. The joint is then fixed, and flexed, and adhesions often form between the opposed surfaces.

In Charcot's disease, on the other hand, the destructive changes involve the superficial parts of the articular surface from the beginning, and quickly spread widely and deeply. Large quantities of cells must be set free into the joints. Effusion is copious and often excessive. The joint movements are increased in range, and irregular; being painless, the patient is not induced to control them, and their jolting character must be productive of many trivial injuries which will maintain the effusion and foster inflammatory changes in the capsule.

Evidently the conditions obtaining in the three affections are highly favourable to dissemination of the cells in Charcot's disease, favourable in osteo-arthritis, and the reverse of favourable in rheumatoid arthritis.

FIG. 249.—The tibia from the same knee as *Fig.* 242, showing the rolled everted edge probably caused by pressure upon softened and rarefied bone. The posterior two-thirds of the upper end of the tibia have been crushed down for some distance so that the part of the upper end seen in the photograph is little more than ⅓ to 1 in. thick (*see Fig.* 250—the opposite knee). (*Specimen* S.C. 88.)

Spurious osteophytic lipping—an appearance, which I believe is peculiar to Charcot's disease—may easily be mistaken for the lipping caused by marginal osteophytes. It is present in a knee (S.C. 88) in which the lower end of the femur is received into a deep cup formed by the anterior part of the head of the tibia, and its crushed down posterior portion blended with numerous capsular osteophytes. The front edge of this cup is thick, rolled, and everted—a condition that has evidently been produced by pressure upon the softened and degenerated bony margin through the play of the femoral condyles within the articulating cavity.

IV. FRACTURES INTO JOINTS.

The influence of the widespread degeneration and softening, and of the intermittent and irregular pressures to which the ends of the bones are subjected, may be far-reaching. It is obvious that the shape of a softened bone may not only be considerably altered, but a bone-end may even be enlarged and expanded owing to the way in which pressure is applied. An important complication of a Charcot's joint, induced by the bony degeneration, is a fracture communicating with the joint. Such fractures are of common occurrence and are usually overlooked. Owing to the absence of pain a patient may continue to use a limb or a joint which is the seat of such a fracture, and even be unaware that anything unusual has happened. The line of fracture in these cases is more or less obliquely vertical, and a considerable fragment is apt to be separated. The fragments may be held in fair apposition by periosteum and by muscular attachments, and firm bony union may result (S.C. 91—1); or they may be pushed apart by the action or shape of the opposed articulating bone; or, again, the fragment may be crushed and lose its natural shape, or be displaced. It is possible that such a sliding, crushing fracture is of slow and gradual production. Thus the remarkable specimen S.C. 88—1 can only be explained by the slipping

FIG. 250.—A Charcot's knee, showing a cup-shaped articulating surface which contains the lower end of the femur—see description in the text. From an old man. *Figs.* 241, 242, 248, 249 are from the same patient.
(*Specimen* S.C. 88—1.)

downwards and backwards of the posterior three-fourths of the head of the tibia, leaving a thin anterior portion to represent the bone. The chief part of the upper end has been crushed obliquely downwards, and has amalgamated with capsular osteophytes to support and contain the lower end of the femur. The condition is symmetrical, yet there is no history of a fracture,

and it is specially mentioned that the man was able to get about. even up-
and down-stairs, till his death (personal communication) (*Fig.* 250).

In another specimen (S.C. 91) the external condyle of a humerus has
been separated, and is incorporated in the capsule more than an inch away
from the humerus. Microscopical examination shows it to be a part of the
original bone, and not a capsular osteophyte.

SUMMARY OF THE ARGUMENT.

The different rheumatoid conditions result from the action and inter-
action of certain important factors.

1. The circulation of toxins is probably the essential and underlying
factor in all four.

2. The measure of the vitality of the joint tissues is shown by their
resistance to the toxic influence. and is the deciding factor in the evolution
of osteo-arthritis and rheumatoid arthritis.

3. A metabolic factor is of decisive importance in gout ; and

4. A nervous factor in Charcot's disease, but in both these conditions
toxins and tissue vitality take their full share.

PRE- AND POST-OPERATIVE TREATMENT OF GALL-BLADDER DISEASE.*

By ARTHUR F. HURST,

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IN recent years it has become a matter of routine in every case of renal disease in which the question of operation is being considered to examine a specimen of urine obtained from the affected kidney by a ureteral catheter and to make a pyelogram. It is a curious fact that of the corresponding examinations required for the investigation of disorders of the gall-bladder only the second is in common use. This is in spite of the fact that, as we have learnt from an experience of over 300 cases in which both methods have been carried out, examination of a specimen of bile obtained with due precautions through a duodenal tube after injection of magnesium sulphate gives information of great value in many cases in which a cholecystogram appears to be perfectly normal or in which its interpretation is too uncertain to be of much use.

The presence of active cholecystitis can be inferred from the discovery of excess of mucus with epithelial débris and sometimes leucocytes. The infecting organism is generally found to be *B. coli*, but occasionally a streptococcus or staphylococcus or *B. typhosus* is isolated. The presence of pigment granules is confirmatory evidence of infection, and cholesterol crystals in the bile indicate that a cholesterol stone is probably present, even if the evidence obtained by cholecystography is completely negative. Lastly, my colleague, Dr. F. A. Knott, has shown that the discovery of a yellow lipoid material is almost conclusive evidence pointing to the presence of a 'strawberry gall-bladder', a point of much help in diagnosis, as this definitely pathological condition is generally associated with a perfectly normal cholecystogram. I should like in passing to add that the value of cholecystography would be much increased if the radiologist would always palpate the visualized gall-bladder, as tenderness localized to the organ is strong evidence in favour of a pathological condition being present even if the shadow appears to be normal. This is of considerable importance, as tenderness found on ordinary palpation in the supposed position of the gall-bladder cannot be assumed to be due to the gall-bladder, which is often displaced, and, on the other hand, a tender gall-bladder in an unusual situation may easily be missed.

The routine investigation of a supposed case of gall-bladder disease should include a test-meal, as achlorhydria is present in 25 per cent of cases of cholecystitis and nearly 50 per cent of gall-stones. Neglect to treat the gastritis, which is generally the cause of the achlorhydria, is likely to lead to

disappointment with the result of the operation, as some of the symptoms supposed to be due to cholecystitis are often the result of the associated gastritis and therefore persist after cholecystectomy.

Pre-operative Treatment.—Many cases of cholecystitis can be cured by means of biliary antiseptics and the stimulation of biliary drainage. I was interested to hear Lord Moynihan state that he knew of no new treatment started within the last few years which had so greatly reduced the necessity for surgical interference in any individual disease as the massive doses of hexamine had done in the case of gall-bladder disease. Many cases which still require operation can be brought to a condition of much greater safety by preliminary treatment of this kind, and I am convinced that a fortnight's medical treatment should invariably precede any operation for chronic disease of the bile-passages. Even in acute and subacute cases the temperature often falls rapidly and the patient's general condition improves very greatly after a few days of treatment with hexamine. With obese patients a longer period of preparation is desirable, both with the object of making the operation less difficult and improving the condition of the patient's heart. It is not difficult by suitable diet to make such a patient lose a stone and a half in weight in a month, during which biliary antisepsis and drainage can also be employed.

It has been known for many years that hexamine is excreted by the bile. but Knott was the first to prove definitely that it acts as a biliary antiseptic in spite of the bile being alkaline, although in the bladder it is inactive unless formalin is set free by the acid urine. Thus pure bile obtained through a duodenal tube has no antiseptic action, but if large doses of hexamine are given, the bile, which can be shown to contain unaltered hexamine, is strongly antiseptic, and any bacteria which were present in it before the treatment was begun disappear. Formerly the maximum dose of hexamine which could be given with safety was limited by the supposed necessity of keeping the urine acid. But hexamine is just as efficient a biliary antiseptic when sufficient alkali is given to make the urine permanently alkaline; under such conditions no formalin is set free and enormous doses can be administered without causing any bladder irritation. For over ten years I have been giving with excellent results 100 gr. of hexamine with 60 gr. of sodium bicarbonate and 60 gr. of sodium citrate after breakfast, after tea, and after a glass of milk or water last thing at night.

At the same time biliary drainage can be promoted in cases uncomplicated by stones by giving on an empty stomach the largest dose of magnesium sulphate which the patient can take without getting diarrhœa. A tablespoonful of olive oil three times a day before meals has the same effect as magnesium sulphate in causing the bile-passages to contract and Oddi's sphincter to relax.

Post-operative Treatment.—The day after operation the hexamine treatment should be recommenced and should be continued for two or three weeks. or still longer if there is reason to believe that the gall-bladder infection was only part of a general infection of the bile-passages. The promotion of biliary drainage by Epsom salts is of even greater importance after than before the operation. and it should be used to the exclusion of all other aperients. Its

continued use after the operation is likely to prevent the symptoms which not uncommonly occur after cholecystectomy for gall-stones and which suggest that a stone has been overlooked in the common bile-duct.   I believe that they are generally due to the disturbance in the neuromuscular mechanism of the biliary passages which follows removal of the gall-bladder and which gives rise to achalasia or less frequently spasm of Oddi's sphincter.

If achlorhydria is present, this can often be cured by treating the gastritis by lavage every morning with dilute hydrogen peroxide (1 drachm to the pint).   If, however, this cannot be carried out, or if the achlorhydria proves to be due to irremediable achylia, 1 to 2 drachms of dilute hydrochloric acid should be given in 5 to 10 oz. of water with orange juice and sugar as a beverage three times a day.

Lastly, it is necessary to say a few words about diet.   It appears to have become customary to give patients definite instructions about restricting their cholesterol intake after operations for gall-stones.   Thus a patient of mine whose gall-bladder was removed at the end of 1930 for gall-stones was advised by the surgeon to take a cholesterol-free diet, with the result that in the following six months she became very weak and lost two and a half stones in weight. She improved rapidly on being given a full diet.   Such restrictions are never necessary.   It is doubtful whether they have any material effect on the percentage of cholesterol in the blood or bile, and moreover when the gall-bladder has been removed, there is no place in which sufficient concentration of the bile can take place for new cholesterol stones to form.   Stones found in the ducts after cholecystectomy were there before ; they never form after the operation.   The only dieting which may be necessary is for associated obesity or gastritis, and no restriction of cholesterol is required.

RECONSTRUCTIVE ŒSOPHAGOPLASTY :

WITH NOTES OF A SUCCESSFUL CASE.

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THE restoration of deglutition after destruction of the thoracic œsophagus is a problem which has exercised the ingenuity of surgeons for many years. While the commonest cause of such destruction is undoubtedly malignant disease, its radical surgical treatment is unfortunately so difficult and has yielded such meagre success that the problem of restoration of deglutition has hardly arisen up to the present time. That earlier diagnosis combined with increased familiarity with the surgery of the posterior mediastinum will eventually bring this disease within the scope of excision seems almost certain.

It is particularly in cases of benign cicatricial stricture that the desirability of restoration is apparent. Such strictures are usually the result of swallowing corrosive fluids (frequently caustic soda) either in error or by design. Unless the stricture is truly impermeable it is probable that gradual dilatation, commencing maybe with the swallowing of a silk thread, will be successful. Plummer[1] and others have reported many cases of successful dilatation in strictures erroneously thought to be complete.

When true impassibility and impermeability are present there can be little hope of relieving the condition by any intra-œsophageal procedure. I believe this stage is indicated when the natural disposal of saliva no longer occurs. So long as anything passes through the stricture one might expect saliva to be retained for a time at any rate in the œsophagus. Progressive hypertrophy and dilatation above the stricture would occur, and, although the contents might be rejected from time to time, the almost continuous expectoration seen in cases of complete stricture would not happen.

Stress must be laid on the importance of establishing the presence of complete occlusion before any reconstruction can be justified, because a review of the literature suggests that in some cases a series of plastic operations which must carry a considerable mortality rate has taken the place of a simpler, safer, but less spectacular form of treatment.

Long before occlusion is complete it is manifest that gastrostomy must have been performed as a life-saving measure. The prospect of a permanent gastrostomy is distasteful in more senses than one. Many of the subjects are young. Apart from the time and trouble entailed in preparing special food and feeding alone and in secret, the unfortunate patient longs for the pleasure and satisfaction of taking food by mouth and swallowing it. The disposal of saliva is another problem not without its embarrassment. It is only when one sees how frequently a receptacle for saliva must be used that one realizes how much is secreted when the œsophagus is obstructed. Roger,

by demonstrating an œsophago-salivary reflex, has explained this increased secretion; but it is probable that in a normal individual there is a continuous flow of saliva and that deglutition occurs much more frequently than is realized. Life must lose much of its attraction to a young man or woman who is bereft of deglutition. Opportunities for social enjoyment are curtailed and there must be a serious handicap to success in any walk of life.

It is not without significance that in all the reported cases of benign fibrous stricture to which I have had access the thoracic portion has been affected. One knows from watching the swallowing of an opaque meal on the X-ray screen that the downward movement is normally retarded or even arrested in the lower half of the œsophagus. It would seem that the rapidity of passage of caustic fluid over the upper half prevents that depth of burning which results in contracting cicatrization lower down. (Mr. C. W. Gordon Bryan has recently informed me that he has a patient with a fibrous stricture of the cervical œsophagus 1½ in. from its commencement.)

The literature of œsophageal surgery reveals that during the present century every possible avenue of approach to the thoracic œsophagus has been attempted. The only record I can find of an intrathoracic operation for benign cicatricial stricture is that of Llobet,[2] of Buenos Aires, in 1900, who performed paravertebral extrapleural mediastinotomy and divided two strictures. The patient died eight days later.

The construction of an extrathoracic conduit which circumvents the stricture and re-establishes continuity between pharynx and stomach has been the aim and ambition of enterprising surgeons since Bircher[3] in 1894 made the first recorded attempt. Both his patients suffered from carcinoma, and died before completion of the work. His method of constructing an antethoracic skin tube has proved the most successful, and is to-day used for the main connection from neck to epigastrium. In both cases death supervened before any attempt could be made to anastomose the upper end of the skin tube to the cervical œsophagus.

Wullstein[4] in 1904, working on the cadaver, was the first to suggest that jejunum might be utilized to connect the stomach with a skin tube. Lexer[5] in 1911 published records of a successful case treated by the Wullstein method. He pointed out that the skin tube had no adverse effect on deglutition, thus disproving Bircher's belief that solid food would not pass down a tube devoid of peristalsis.

In 1925 Rovsing[6] described a method similar to that of Bircher. The skin tube was extended downwards to include the gastrostomy without the intervention of any other structure. He published records of two successful cases. Mention must be made of Jianu's[7] method modified later by Rutkowski[8] whereby the greater curvature of the stomach is utilized to act as the connecting channel with the front of the chest. Dengel[9] has recently reported a successful case treated by this method. Three years and three months elapsed between the first operation and final healing. I cannot help feeling that time would have been saved had the skin tube been constructed at the first operation.

It would not serve any useful purpose to enumerate every attempt that has been made to utilize the various portions of the alimentary tract to bridge

the gap between stomach and cervical œsophagus. Many failures have demonstrated the futility of expecting any alimentary viscus to retain its blood-supply when stretched up to the episternal notch. General agreement has been reached that a skin tube is necessary for at least the antesternal region. Whether this be continued downwards to include an ordinary gastrostomy as in Bircher's and Rovsing's methods, or be continued as jejunum or stomach flap is a matter of individual choice. By far the largest number of successes have attended the use of a skin tube with jejunum as the connecting link. It is this method that I used.

I would urge the advisability of constructing the skin tube first, even though the length required may be difficult to forecast. In the absence of saliva and gastric or jejunal secretion it should not be difficult to ensure primary union of the skin plastic. Although the effective anastomosis of the upper and lower ends of the skin tube with œsophagus and jejunum will still remain the most precarious undertaking, it must be manifest that absence of scarring and recent infection will increase the likelihood of success.

CASE REPORT.

V. B., female, aged 27 years.

SUMMARY.—

May, 1927.—Swallowed ammonia. Onset of dysphagia two months later.

September, 1927.—Unable to swallow solids or liquids. Two dilatations through œsophagoscope produced only temporary improvement.

October, 1927.—Gastrostomy.

February, 1928.—Two further attempts at dilatation restored some swallowing of liquids.

April, 1928.—Unable to swallow saliva. Stricture divided by diathermy. Subsequent development of mediastinitis and broncho-œsophageal fistula.

January, 1929.—Three further attempts at dilatation failed.

February, 1929.—Reconstruction of œsophagus commenced. (1) Antesternal skin tube fashioned. (2) Cervical œsophagus anastomosed to upper end of skin tube.

April, 1929.—(3) Jejunal loop inserted between lower end of skin tube and stomach.

June, 1929.—Unsuccessful attempt to close fistula at upper end of jejunum.

September, 1929.—Second attempt to close fistula.

December, 1929.—Final healing.

HISTORY.—The patient was a female, 27 years of age. In May, 1927, after disappointment in a love affair she attempted to commit suicide by drinking ammonia. After treatment at the General Hospital, Birmingham, she was transferred to the Infirmary, where she remained for one month, and apparently made a satisfactory recovery. Dysphagia was first noticed in July of the same year, firstly with solid food. In September she was admitted to the General Hospital under my colleague, Mr. Woodman, as the dysphagia had become almost complete. Œsophagoscopy then revealed a stricture at the level of the 5th dorsal vertebra; free bleeding occurred from active

ulceration. The stricture was entered for 2½ in., but was not traversed; dilatation was found to be impossible. An X-ray at the time demonstrated the stricture. A week later œsophagoscopy was repeated. The upper end of the stricture admitted a No. 10 filiform bougie; active hæmorrhage occurred and further dilatation was not considered safe. Subsequently swallowing was impossible for twenty-four hours. Egg flip was then swallowed with difficulty for two days, when suddenly deglutition became impossible. The weight was now 5 st. 12 lb. Gastrostomy by Senn's method was carried out by my Assistant Surgeon, Mr. Scott Mason. The stomach was reported to be small but with healthy mucous membrane. By February, 1928, the weight had increased to 7 st. 5 lb.

FIG. 251.—Antero-posterior view showing stricture, and barium passing into left bronchus.

In February, 1928, Mr. Woodman again performed œsophagoscopy.

A portion of the stricture was removed for pathological examination. It was found to consist of granulation tissue with abundant cell infiltration. A bougie was passed on that occasion and again a week later. The patient left hospital at the end of February able to swallow a little, but a week later no solid food could be taken, and liquid only with difficulty.

On April 1 she was re-admitted to hospital unable to swallow anything, even saliva. Œsophagoscopy by Mr. Woodman demonstrated a stricture 2½ in. long. A bougie was passed and the opening enlarged by diathermy. Some pyrexia followed, but five days later she swallowed a cup of tea. Radiographic examination now showed a broncho-œsophageal fistula just above the stricture (Fig. 251).

By December, 1928, she was unable to swallow saliva, and feeding was carried out entirely by the gastrostomy.

Œsophagoscopy by Mr. Adams now showed a stricture 10 in. from the incisor teeth which bled freely and was impassable to any bougie. Three days later a filiform bougie was inserted and dilatation commenced, but deglutition was not restored and it was decided that such treatment would not be successful.

On Feb. 8, 1929, she was transferred to my ward at the General Hospital. Twenty-one months had then elapsed since swallowing the ammonia. She

carried about with her a bottle into which she expectorated every minute or less. The general condition was good. The patient readily consented to submit to any procedure which would restore deglutition.

OPERATIONS.—My first operation was done on Feb. 14, 1929. It had for its object the formation of a subcutaneous skin tube to extend from the upper to the lower aperture of the thorax. Two parallel longitudinal incisions 2½ in. apart were made from the suprasternal notch to below the ensiform cartilage. The right-hand edge of the strip of skin was undermined for 1 in., the left-hand edge for ½ in. The two edges were then rolled over and united to each other by interrupted stitches of very fine silk, passing through cuticle only and with the knots lying on the inside of the tube. The suture line was reinforced with fine interrupted catgut sutures. The lateral edges were then undermined and flaps mobilized so that they could be approximated and united with sutures. It was difficult to avoid tension at the upper and lower ends. Unequal undermining of the sides of the strip had succeeded in preventing the two lines of sutures being superimposed. It also brought the skin tube to the left of the mid-line. Satisfactory healing by first intention ensued. (*Fig. 252.*)

Fourteen days later the second stage of the operation was undertaken. It was witnessed by members of the Otolaryngological Section of the Royal Society of Medicine who were then visiting Birmingham.

The cervical œsophagus was exposed through a longitudinal incision extending upwards from the upper end of the skin tube at the inner end of the left clavicle. The recurrent laryngeal nerve was displaced medially. The œsophagus was isolated well into the superior mediastinum and divided as low as possible between cholecystectomy forceps. The lower end was closed and invaginated. After preparation of the edges of the upper end of the skin tube a large rubber catheter was passed up the skin tube and made to enter the upper section of the œsophagus. The mucous membrane of the latter was now sutured to the cuticle of the skin tube around the catheter, using the same fine silk sutures with all knots in the lumen; oversewing with catgut stitches completed the anastomosis. A flap of skin on the left side of the neck was mobilized and swung down to cover the raw surface. A small rubber drainage tube was inserted down to the closed lower section of the œsophagus.

Subsequently there was some leakage of saliva from the suture line, with consequent local wound infection. Attachment of a suction pump to the
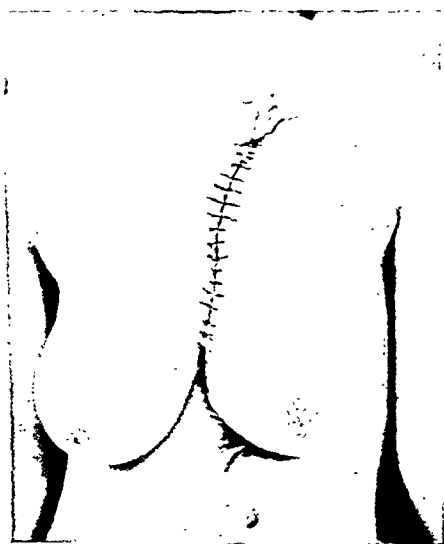
FIG. 252.—Photograph showing completion of first stage of operation. The gastrostomy opening is shown below the lower opening of the skin tube.

lower end of the catheter lying in the skin tube did not abolish the leakage. Both the drainage tube and the catheter were discarded within two weeks of the operation. Saliva was then escaping from the lower end of the skin tube and from a fistula at the anastomosis.

Four weeks after the operation a catheter was passed successfully from the mouth through the anastomosis into the skin tube. Leakage from the fistula diminished gradually. On April 2 a No. 18 stomach tube was passed through the mouth past the junction, and on April 6 the same instrument was pushed completely through the lower end of the skin tube.

On April 10, the stomach having been washed out with sodium bicarbonate solution, a cystoscope was passed through the gastrostomy opening. The main object was to ascertain whether there was sufficient stomach to the left of the gastrostomy to permit anastomosis with jejunum. The gastric mucous membrane was seen to be healthy ; there were no signs of scarring or contraction. It was noticed that the introduction of sodium bicarbonate solution caused opening of the pyloric sphincter.

On the following day gastrostomy was repeated ; an attempt to pass a ureteric catheter through the cardiac opening of the œsophagus failed.

By April 16 a No. 22 stomach tube could be passed from the mouth and drawn completely through the epigastric opening.

On the following day the third stage of the operation took place. Through a left pararectal incision on the outer side of the gastrostomy a portion of upper jejunum 6 in. in length was selected with a maximum length of mesentery and a separate sheaf of vessels from the posterior abdominal wall. This portion was isolated so that only its vascular supply remained. Continuity of the jejunum was re-established by end-to-end anastomosis and approximation of the mesenteric edges. The isolated jejunal loop was brought upwards through the transverse mesocolon and the anterior layer of the great omentum to the front of the stomach, into which its distal end was sutured. The proximal end of jejunum was brought through the upper angle of the abdominal wound into the subcutaneous layer, where it was united to the lower end of the skin tube by the technique already described. Great care had to be taken to avoid tension on the vascular pedicle. This did not permit easy approximation of the antimesenteric edge of jejunum to the anterior edge of the skin tube. Here some of the sutures tore through the bowel. Undermining of the skin enabled all raw surfaces to be covered in and no drainage was employed.

After nine days of satisfactory progress a drink of water was given. Some leakage occurred through both suture lines. Ten days later a No. 12 stomach tube was passed into the jejunal loop, and on May 21 the same tube was passed into the stomach. By May 30 the patient was drinking fluids freely. The fistulæ were covered by pads and strapping. Leakage was not enough to be troublesome.

In June an unsuccessful attempt was made to close the lower fistula by a small plastic operation. The patient left hospital in July taking all her food by mouth and with the upper fistula healed ; pressure by hand prevented leakage from the lower fistula. In September she was re-admitted for another plastic operation to close the fistula. It was only partially successful. Later

the edges were cauterized, and by December healing was complete. The gastrostomy opening closed spontaneously.

SUBSEQUENT HISTORY.—Shortly after discharge from hospital the patient returned with a lump of meat impacted at the upper skin junction; within an hour it had passed on without treatment; no further difficulty has occurred. She leads a perfectly normal life, eating and swallowing ordinary food (*Fig.* 253). Her weight is now 8 st. 1½ lb. During deglutition the bolus can be seen to descend the skin tube rapidly. It is then carried on into the stomach by visible peristalsis of the jejunal loop. That gravity is not necessary to convey food through the skin tube was demonstrated by the successful swallowing of barium paste when she was lying in an inverted position (*Fig.* 254). The radiographic screen showed that the heavy opaque meal quickly passed into the stomach, its passage being hastened by repeated swallowing of air (*Figs.* 254–256). Dr. Newton, of Moseley, suggested that deglutition was accomplished by air pressure.

FIG. 253.—Photograph showing patient drinking. Active peristalsis is visible in the subcutaneous loop of jejunum.

FIG. 254.—Lateral view of new œsophagus with the patient inverted, showing barium passing through upper skin junction and air-bubble in front of sternum.

and this may be the explanation of the rapid descent of food without the aid of œsophageal peristalsis. The patient has not vomited since her first operation. Whether vomiting could occur without reverse peristalsis of the jejunal loop is an interesting speculation.

I desire to record my acknowledgements to Professor Zaaijer, whose successful case at Leyden was my inspiration; to Mr. Woodman, who provided me with the opportunity; to my House Surgeons, especially Mr. Knight and Dr. Taylor, for their valuable help; and to Professor Lockhart for assistance with the illustrations.

FIG. 255.—Lateral view of new œsophagus showing barium passing through lower skin junction and jejunum into the stomach.

FIG. 256.—Antero-posterior view of same state of affairs as in *Fig.* 255.

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BIPARTITE CARPAL NAVICULAR BONE.

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THOUGH the occurrence of congenital bipartite navicular bone is referred to in surgical writings,[2, 3, 4, 9] the condition appears to be rarely found and its existence sometimes forgotten altogether.

A divided navicular bone was found in the right carpus of a male dissecting-room subject. The bone was divided about its middle, the line of division passing from the articular surface for the radius, near its lateral

FIG. 257.—Dorsal aspect of dissected carpi. Left and right.

edge, to the middle of the concavity for the head of the os capitatum, so that there was a larger radial portion, distally situated, articulating with the ossa multangula, and a smaller ulnar portion articulating with the os lunatum.
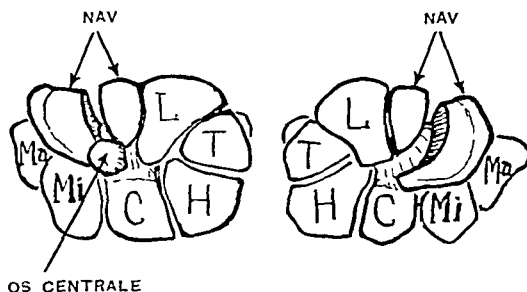
FIG. 258.—Diagram of Fig. 257. L, Lunate; T, Triangular; H, Hamate; C, Capitate; Mi, Lesser multangular; Ma, Greater multangular.

This is the usual site of division. Rarely the bone is divided into dorsal and palmar portions.[1, 6] The opposing surfaces of the two portions were clearly defined and covered by a dense layer of eburnated bone without any cartilage apparent to the naked eye. The cavity between them was separated from the cavity of the radiocarpal joint by a ligament uniting their proximal edges.

In the discussion which arose among my colleagues as to whether this was a fracture or a congenital division of the bone, it was suggested that the other carpus be examined.   It was found that the latter had been dissected two years previously and preserved in a jar—hence the discoloration evident in the photograph (*Fig.* 257).   It presented a precisely similar appearance to the right carpus, with the additional presence of an isolated bony nodule attached to the dorsal aspect of the radial portion of the navicular bone
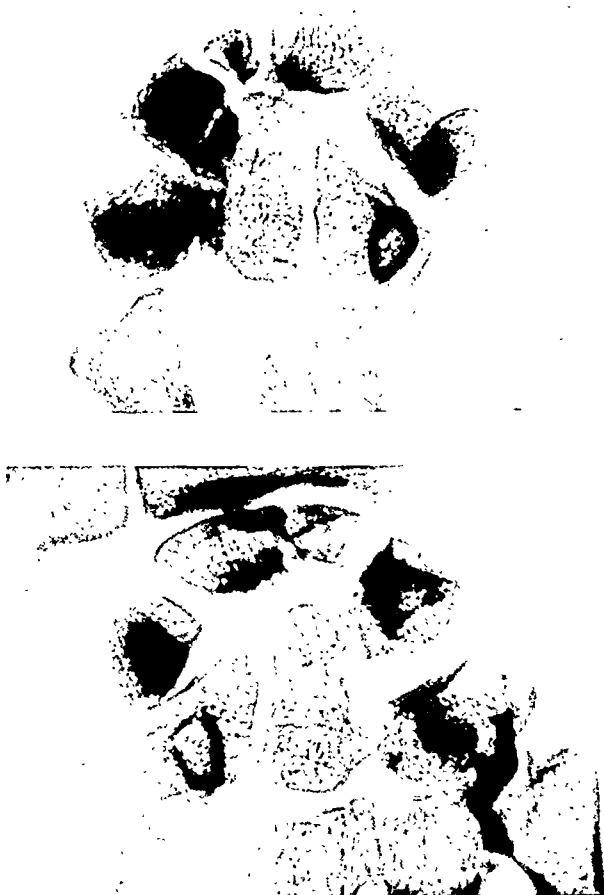
FIG. 259. —Radiogram of left and right carpi, taken after dissection, so that joint spaces are opened up.

where it articulated with the capitate (*Fig.* 258).   The radiogram (*Fig.* 259) demonstrates the well-defined adjacent edges characteristic of bipartite navicular bone.

The navicular bone is usually described as ossifying from a single centre about the sixth year.   Pfitzner[13] has shown that it is formed of two main elements, a radial and an ulnar, and two accessory bones—the os radiale

externum, which is incorporated in the tuberosity but may be separate.[7] and the os centrale, which may, however, fuse with the capitate or remain as a separate ossicle on the dorsum of the carpus. A double cartilaginous centre has been found by Bardeleben, Thilenius, Rambault, and Renault (cited by Wolff[18]), though other writers have reported negative results.

Dwight[3] has found the bone subdivided at birth. The same author states that in a considerable number of cases, probably 2 per cent, there are signs of an incomplete division, or, more accurately, of an incomplete union of two originally distinct elements. He describes a post-mortem specimen of a divided navicular bone of the right hand in which the opposing surfaces were in perfect apposition and permitted no movement. Microscopical examination confirmed the presence of a cartilaginous coating.

It is admitted that the majority of specimens have a pathological appearance, with little or no intervening cartilage, and polished, eburnated, opposing surfaces, possibly because they are not anatomically adapted to form a good functional joint. This may be accompanied by arthritic changes in the other carpal bones. It may thus be difficult to distinguish a congenital divided navicular bone from an old fracture with non-union and subsequent osteo-arthritis. Indeed, the cases reported by Struthers[15] and Johnson[8] were ascribed to fracture.

The case described by Leboucq, cited by Gruber,[6] was bilateral. In several instances, and in the case now described, a supernumerary carpal bone was present, especially an os centrale.[6, 8, 17] In some cases it might be regarded as abnormal ossification occurring in the dorsal ligaments as a result of faulty mechanics, but in the present instance the position of the bone and its clear demarcation from the surrounding ligaments suggest that it is probably an os centrale.

Most of the reported specimens of congenital bipartite navicular bone have been found in the cadaver. Pfitzner found the condition unilaterally 9 times in 1456 wrists, but it is contended that some of these cases may have been fractures. Gruber[6] examined 3007 navicular bones, of which 4 were bipartite and 1 was tripartite, but in the next 1000 only 1 was divided, and this indefinitely.

Codman,[2] in looking through 1040 X-rays of wrists, did not find any divided scaphoid which was not definitely associated with injury " (except for three cases which he considered to be fractures on account of the marked osteo-arthritic changes present). Köhler[10] has not seen an os naviculare bipartitum in an X-ray, and " agrees with Wolff in supposing that the majority of the cases of naviculare bipartitum are simply pseudo-arthroses following upon fractures."

Schulz[14] records a case of bilateral congenital bipartite navicular bone which was revealed by a radiogram. There was no history of injury, but the patient complained of pain in the wrists which began at the age of fifteen. Mouchet[12] describes a case of congenital bipartite navicular bone in the left wrist of a young man who had developmental shortness of the phalanges. There had never been an injury and the condition was diagnosed by X rays. Faulkner[4] quotes a case with no history of any trauma which Blencke diagnosed by X rays. He himself describes a case of gonorrhœal arthritis occurring in the joint between the two portions of a divided navicular bone.

The condition was bilateral and there was an interval of a few months between the onset of symptoms on the two sides. Radiograms were taken and both sides were subsequently operated on. Naked-eye examination showed irregular surfaces coated with degenerated cartilage.

McKendrick[11] does not mention congenital bipartite navicular, but states, " it is no uncommon thing to find a fractured navicular when X-raying the wrist for conditions other than injury." He ascribes such cases to an old fracture which has been forgotten by the patient, but it is just as likely that they are cases of bipartite navicular. Indeed, McKendrick himself states that it takes considerable violence to cause fracture of the bone. Todd[16] discusses the differential diagnosis between congenital division and fracture of the body of the navicular bone. He points out that the violence necessary to break it is not likely to be forgotten and that the history is definite and characteristic. A recent case of fracture shows unmistakable physical signs which are usually diagnostic of the condition without the help of X rays.

SUMMARY.

There appears to be good evidence for the occasional occurrence of a congenital bipartite navicular bone, probably about once in a thousand times.

It is to be recognized : first, by the absence of a definite history of injury ; secondly, by the meagreness of the physical signs ; and, thirdly, by the presence in an X-ray picture of a thin well-defined and continuous line produced by the layer of compact bone covering the spongy bone. In the case of a fracture, a broader, less sharply defined line may be expected, which may show irregularities in width. Fourthly, a similar appearance, if present in the other wrist, is strong confirmatory evidence, but the presence of a normal navicular on the other side does not invalidate the diagnosis of congenital origin.

The condition becomes of special importance in assessing claims for compensation on account of an alleged injury.

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EIGHT LETTERS OF JOSEPH (LORD) LISTER TO WILLIAM SHARPEY.

(Concluded from p. 164.)

By C. R. RUDOLF.

11, Rutland Street.
Edinburgh,
26 *Oct.* /59.

My dear Dr. Sharpey,

Agnes wrote to you the day before yesterday enclosing my testimonials, and mentioning, I believe, the circumstances which induced me to send in my application to the Home Office. Of that application I received an acknowledgement from a clerk on Sir G. C. Lewis'[19] behalf; and I also know that the Lord Advocate has received his copy of the testimonials : so that I am now fairly in the field. And yet it seems again quite uncertain whether Lawrie has really resigned : at all events, if he has, neither the Glasgow Professors nor the Lord Advocate know anything of the matter. And yet I am well satisfied that I have sent in my application. Benjamin Bell,[20] who is kindly interested on my behalf, has seen the Lord Advocate, who told him that when he was in London (some months ago) Fergusson had pressed him very hard regarding Edwards, but he seemed anxious to have a further conversation with Mr. Bell, and also agreed to act on his suggestion that he (the Lord Advocate) should also refer to Allen Thomson and Douglas Maclagan. The latter is now fully on my side, Spence having retired from the field, and has great influence with Moncreiff, and, withal, is President of the College of Surgeons this year. Benjamin Bell says the Lord Advocate seemed quite open to conviction, and promised to read my testimonials carefully. So things look pretty favorable in that quarter.

Also Sir James Clark[21] called on Mr. Syme to-day, and had a conversation with him on the Glasgow business, and seems quite clear as to the superiority of my claims. I mentioned to Sir James (when I saw him for a minute or two) that I had stated in my letter of application to the Home Secretary that Sir James was willing to act as referee; and he quite approved. Whether or not he may be referred to is, however, quite uncertain, and I rather expect, from Sir James saying that " it would come best in that way ", that he will not address the Home Secretary unless he be applied to. I have sent copies of the testimonials to the Glasgow Members of Parliament and also to the Professors, but beyond this I am keeping pretty quiet till the vacancy is distinctly declared. I have gone on talking about my own affairs in a way that would be hardly pardonable did I not know that you are interested in it.

And now my sheet being full, I had better conclude, remaining with kind regards from Agnes to you,

Yours ever most sincerely,
Joseph Lister.

W. Sharpey, M.D., Sec. R.S.

2, Rutland Street,
Edinburgh,
27th March /64.

My dear Dr. Sharpey,

Happening to be here at the time your letter to Mr. Syme arrived, I had the great pleasure of learning that you were the author of the remarks on Medical Education in this week's Lancet, and therefore that you approve of the plan of substituting class examinations for final examinations of degrees and licences in the Scotch schools. In Glasgow the Medical Faculty have unanimously agreed to adopt this principle, and the Senators at their last meeting acquiesced in the proposal. So we shall begin to work the new system this session, and I am sure it will prove an immense boon to the better class of our students. It will both act as a powerful incentive to work at each subject earnestly while they are engaged in it, and also leave their minds free to engage in the work by taking off from their shoulders the heavy load of the " cramming system ". I was at one time afraid that the Assessors would have more work thrown upon them than they could accomplish; but it occurred to me that all that would be necessary would be for each Professor, when he has himself ascertained which of his students have exhibited sufficient attainment, to shew to an Assessor the written answers of these good students only. These will probably not be more than about a quarter or a third of the class, if so many, and thus the Assessor's labour will be greatly lightened. We are accordingly intending to act in this way: and it is a very fortunate thing that, thanks to the advice you gave me (which advice as coming from you was I believe a main means of enabling me to introduce the thing) we in Glasgow have for the last three years given our students class certificates of four different orders of merit. All that will be necessary will therefore be for the Assessors to look over the answers of those whom we think deserving of first and second class certificates, and then it will come to be understood that the receipt of such a certificate by a student exempts him from further examination. I believe that this system will so lighten the students' labour that any reduction of the curriculum will be quite unnecessary. For example, take even Botany. I think it will be anything but a disadvantage to a man to attend a course of Botany in the summer session of his first year, acquiring practical knowledge of plants by the excursions, etc., and learning habits of observation; provided that at the end of this course he can feel that the subject need not trouble him further. I concur most cordially in what you say about the essential thing being to get a man to work really well at a subject once.

The appointment of the Assessors was also a most happy thing for us. Without them we could not have worked this new plan of examination : which in truth will exercise a scarcely less beneficial effect upon the examiners than upon the students. For some of our Professors, I am sorry to say, did the examining duty in a very imperfect slovenly way, so that a student who only deserved a 4th class certificate might get a 1st and vice versa ; which not only caused dissatisfaction in times past, but would have proved fatal to the proper working of the new system. Now, however, the Professors will take very good care that they do not send up to the Assessors answers from their

students of which they ought to be ashamed. And indirectly a good effect will also be produced upon our teaching, for it will not do for us to give hobbies too prominent a place or omit other matters of essential moment. I cannot tell you the happiness I feel in looking forward to this plan. I have been deeply impressed with the evil of having the student's mind occupied merely with a sort of unhealthy stuffing of the memory, to the utter neglect, if not destruction of the judgment, which is, I think, the most important faculty for a medical man. I think in Glasgow there has been a peculiar tendency to this : the very diligence of the students seeming often almost mischievous, from the slavish way in which they learnt what they were taught. And yet the poor fellows had no leisure for anything else.

With regard to the practical examination I must say I do not quite agree with you. As to anatomy there is no question of the immense advantage any more than of the practicability of the mode of examining, and Mr. Syme too is far from objecting to it there. But the case of Medicine and Surgery is altogether different. Letting alone the question of the propriety of using patients for the purposes of examination and (especially at a large school such as ours, where we expect 60 candidates to come before us at once on the next occasion) the practical difficulties in the way of carrying out the thing, the grand objection seems to me to be that you *cannot* expect students to be *practitioners* as they are *anatomists*. The best proof of this is that both in Edinburgh and Glasgow it is our *graduates*, and the very best among them, who take the offices of Residents in the Infirmary for the very purpose of doing that with reference to practical Medicine and Surgery which the student in the dissecting room does with reference to practical Anatomy. To be able to diagnose a case and decide at once upon the treatment requires a very high degree of practical attainment. I know for my own part, it was more by good luck than anything else that I was able to make anything like a decent appearance at the practical examination for M.B. at the London University. I never acquired anything *approaching* the sort of style that this examination professes to test till I came to be Dr. Walshe's assistant *after* my graduation. It may be said that it is very desirable to do anything you can to encourage practical work at the cases. But I believe that if the student is relieved from the labour of cramming and at liberty to look at his studies in some other light than merely as means for getting him through his examination, he will see the obvious importance of hospital work, and regard the clinical informa-tion he gains as having its own sufficient reward : being in fact the principle stock in trade for his future practice. Thus I confidently expect that under our new system hospital study will flourish with a healthy vigour far superior to that which could be fostered by any Clinical examinations : and I only wish I could clearly see a way in which the same system could be introduced in London.

I did not expect to have troubled you with so lengthy an epistle, which I only hope you will not think presumptuous, but believe that I remain as ever,

Yours with the greatest respect and gratitude,

JOSEPH LISTER.

W. SHARPEY, Esqr., M.D., Sec. R.S.

THE REPORTS OF THE TWO REFEREES (SIR) JAMES PAGET AND JOHN GOODSIR IN THE POSSESSION OF THE ROYAL SOCIETY.

REPORT ON MR. LISTER'S PAPER ON THE EARLY STAGES OF INFLAMMATION.

This communication consists of an introduction, four sections and two supplements, each of which requires separate notice.

The introduction contains some observations on the early phenomena of inflammation, intended chiefly to prove their similarity in warm and in cold-blooded animals : and an account of the theories of previous writers respecting the stagnation of blood in an inflamed part. Some of the observations, though simple, are I believe novel as well as instructive and should be published. The account of the theories may safely be omitted (pp. 6–10).

The first section " On the aggregation of the Corpuscles of the Blood " seems to me all worthy of publication.

The second " On the Structure and Function of the Blood-vessels " would be improved, I think, by the omission of all the discussion of vital affinities, etc., contained in pp. 22–25. The author appears to have laboured under the influence of recent discussions in Edinburgh, which, in both this and other parts of the paper has led him to give unnecessary extent to comparatively trivial and uncertain things. The rest of this section is valuable and necessary to the understanding of the parts following it.

The third section " on the effects of irritants upon the circulation " deserves publication entirely.

The experiments are simple and instructive—they confirm, with more evidence than has yet been published, the view that the stagnation of blood in the living vessels is not dependent on changes of size of the vessels, but, probably in molecular or vital changes in their walls, or in the tissues external to them.

The fourth section is the most valuable of all—the observations on the pigment cells of the frog's web give the best or only visible proof yet discovered, that in the early stages of inflammation in vascular parts, the tissues external to the blood-vessels are independently and even primarily affected by the stimulus applied. The whole of this section should, I think, be published, but I would suggest that the expressions about " paralysing " the attractive and repulsive, or the concentrating and diffusing, forces in the pigment cells imply much more than is proved and might be advantageously changed.

The concluding remarks, pp. 63–68, which seem to have been written under the influence already referred to, may be safely omitted.

Of the two supplements, the first, " On the influence of the nervous system on the arteries " should be published, if at all as a separate paper. And I should certainly recommend it for publication, but that I think the author could much increase the value, by increasing the precision of his results, if he would continue and extend his observations—. At present he has not attained to much more than the proof that the lower part of the spinal cord in the frog exercises an influence on the blood-vessels in the hind

feet similar to that proved by M. Brown-Sequard and others for the spinal cords of mammalia—. It would be interesting to imitate M. Brown-Sequard's experiments on sections of one lateral half of the cord, and to contrast the states of the vessels in the two webs : and by this and other experiments, I think, the author would discover more precisely than he has yet done. the seat of the nervous centre of the blood vessels. I would suggest. therefore. that instead of publishing this supplement in the Transactions, the communicator of the paper should be requested to recommend the author to extend this part of his enquiries.

The second supplement " On the Pigmentary System of the Frog " should, I think. be published entire. and as a separate paper, preceding the essay on inflammation.

<div align="right">JAMES PAGET.</div>

July 30, 1857.

<div align="center">" Mr. Paget's Report on Mr. Lister's Paper "
July 30, 1857.</div>

<div align="center">THE COMMITTEE OF PAPERS, ROYAL SOCIETY.</div>

GENTLEMEN.

Having been requested by you to give my opinion regarding the eligibility of Mr. Lister's " Paper on the Early Stages of Inflammation " for publication in the Philosophical Transactions, I beg to state that while this paper contains observations and results, which, if verified, will have an important influence on the progress of the subject of which it treats, it would at the same time. in my opinion, as a paper on a pathological subject, be more suitable for the Philosophical Transactions if it had been more condensed, especially in the 'Introduction' and 'Concluding Remarks'.

<div align="center">I am. Gentlemen,
Your obed serv^t,
JOHN GOODSIR.</div>

<div align="center">University of Edinburgh.
Oct. 4th, 1857.</div>

<div align="center">COMMENTARY.</div>

From the time that Lister as a medical student came under the influence of William Sharpey—the first Professor of Physiology at University College —he became his devoted friend and admirer. Under his tutelage was initiated a love of research that was to lead him from his early inquiries in microscopical science and the processes of inflammation to the culminating labours of his antiseptic treatment. It is on Sharpey's advice and with his introduction to James Syme that Lister arrives in Edinburgh, where the first phase of his career was passed and from which place these letters were written. In this correspondence through a number of years—physiology. the Glasgow election. medical education—everything is submitted with deference to his teacher and friend for whom affection and respect are everywhere apparent.

The first, second, third, and sixth of these letters deal primarily with the subject matter of his paper on "The Early Stages of Inflammation",[22] and its two supplements, which, as Lister was not yet a Fellow, were 'communicated' by Sharpey, their Secretary, to the Royal Society. They are also concerned with the criticisms of (Sir) James Paget and John Goodsir, the two referees chosen to decide on the suitability of the paper for inclusion in the *Philosophical Transactions*. The original MSS. is unfortunately lost, but the reports of the two referees are available and are reprinted above. Alterations in format, with the publication of the two supplements, each as a separate paper,[23] are adopted ; but Lister is at issue with his critics on the question of 'vital affinities of the blood' and the physiology of the pigment cell. That these 'vital affinities' did, as he suggests, considerably exercise medical opinion at that time is supported by evidence at home and abroad. Alison[24] approves of the theory, and foreign physiologists, including Haller, Magendie, Kaltenbrunner, et al., contribute their observations to the contemporary press. Lister, however, is more concerned with the objection to his term 'paralysis' in relation to the concentrating and diffusive forces of the pigment cell. Writing to his father he says, " I felt it therefore to be by no means agreeable to have the correctness of the expression 'paralysis' questioned by Paget and Sharpey ", supplying him also with a résumé of the experiments with which he sought to convince Sharpey. He successfully met both objections, and his views as originally expressed were ultimately incorporated in the *Philosophical Transactions*.[25]

Letters 4 and 5, dated June 28 and July 7, 1858, were the forerunners of his paper, " Preliminary Account of an Inquiry into the Functions of the Visceral Nerves, with Special Reference to the So-called Inhibitory System ",[26] which appeared in the *Proceedings* of the Royal Society as a letter to Dr. Sharpey. In this form submission to referees could be dispensed with. There were others in the field and he was anxious to get his views into print, and but little more than two months were allowed to elapse between his first observations and the completed paper. To-day publication in the *Proceedings* is more sought after, but in the middle of the nineteenth century the *Philosophical Transactions* took precedence, its pages being reserved for original work of exceptional importance. It may, therefore, be conjectured that this paper of Lister's was not considered of sufficient moment to warrant, as he wished, its inclusion.

The concluding months of 1859 see Lister's stay in Edinburgh drawing to a close. In August he writes to his father[27] that Dr. Laurie is resigning his professorship in the University of Glasgow for reasons of health, and discusses his own possible candidature. By Oct. 12, it will be seen, he had definitely entered the field from which he emerged the chosen candidate early in the following year.

In 1864 the question of medical education was much to the fore. In Scotland, Syme and Lister led the way, and the Senates of Edinburgh and Glasgow Universities had already reorganized their system of teaching. In London proposals by Syme and Christison for modification of existing practice on similar lines were under consideration by the Medical Council. A series of articles on medical education appear in the *Lancet*,[28] where the

proposed innovations are critically examined. A note of antagonism by no means unusual in the polemics of the time is discernible in its Editorial wherein London teachers are exhorted to formulate their views before the assembling of the Council, whilst fears are entertained that " both the number and the decision of Scotch, Irish and University Members of the Council will give them a great advantage in enforcing their views." In the last of our letters Lister, least combative of men, deals with Sharpey's contribution to the *Lancet* with his usual serenity and good sense.

The original Lister letters are preserved in the Library of the Royal College of Surgeons.

I should like to acknowledge my indebtedness to Sir Arthur Keith for his kind advice, to Sir Edward Sharpey-Schafer for his note on Miss Colville, to Sir D'Arcy Power for his invaluable help in preparing these letters for the press, and to the Royal Society for permission to print the reports of Sir James Paget and John Goodsir.

NOTES.

[1] GEORGE GABRIEL STOKES. Secretary of Royal Society from 1854 to 1885.

[2] SIR JAMES PAGET, 1814–99. Author of *Surgical Pathology*. 1853.

[3] JOHN GOODSIR, 1814–67. Professor of Anatomy at Edinburgh University.

[4] WILLIAM BENJAMIN CARPENTER, 1813–85. Author of *Principles of General and Comparative Physiology*.

[5] EMIL WILHELM, RITTER VON BRÜCKE. 1819–95. Professor of Physiology at Vienna, 1849.

[6] WILHELM VON WITTICH. 1821–82. Director of Physiological Institute at Bonn. Worked with Helmholtz.

[7] EMIL HARLESS. 1820–62. Writes from Erlanger on the influence of gases on the blood, 1846.

[8] EDUARD FRIEDRICH WILHELM PFLÜGER, 1829–1910. Director of Physiological Institute at Bonn.

[9] AUGUSTUS VOLNEY WALLER, 1816–71. Assistant of J. L. Budge (*see below*). Professor of Physiology, Birmingham.

[10] JULIUS LUDWIG BUDGE (Senr.), 1811–88. Published his paper on the movement of the iris at Brunswick, 1855.

[11] TUFFER WEST. Well-known engraver of wood blocks : the business is still carried on by his successors.

[12] THOMAS WHARTON JONES, 1808–91. Experimental physiologist. Professor of Ophthalmology at University College.

[13] MISS COLVILLE. Sir Edward Sharpey-Schafer writes : "Miss Colville was Dr. Sharpey's niece and kept house for him for a time at Hampstead, but she fell ill and died, 1 am not sure when. At any rate, as a result, he gave up house-keeping and went to live in lodgings in Torrington Square. She had a brother in either the Indian or Colonial Medical Service, Major Colville, who was stationed for many years at the Consulate at Baghdad and to whom I believe Sharpey left the greater part of his estate : but he did not long survive to enjoy it. He married after his retirement somewhat late in life. I know very well what a high opinion Lister always had of Sharpey."

[14] LAZZARO SPALLANZANI. 1727–97. Distinguished naturalist. Author of *Observations on the Circulation*. 1777.

[15] AGNES, elder daughter of James Syme and wife of Lister.

[16] OTTO SPIEGELBERG, 1830–81. Professor of Midwifery at Göttingen.

[17] GEORG MEISSNER, 1829–1905. Professor of Anatomy and Physiology at Basel.

[18] MORITZ SCHIFF, 1823–96. Professor of Physiology at Basel.

[19] SIR GEORGE CORNWALL LEWIS, BART., Home Secretary, 1859–61.

[20] BENJAMIN BELL, 1749–1860. Surgeon to the Edinburgh Royal Infirmary. Not related to Sir Charles Bell.

[21] SIR JAMES CLARK, 1788–1870. Physician-in-Ordinary to Queen Victoria, Physician to the Duke of Kent.

[22] "On the Early Stages of Inflammation ", *Philosophical Transactions*, Part 2 for 1858, p. 645. (Read June 18. 1857.)

[23] " An Enquiry regarding the Parts of the Nervous System which regulate the Contractions of the Arteries ", *Philosophical Transactions*, Part 2, for 1858, p. 607. (Read June 18. 1857.)   " On the Cutaneous Pigmentary System of the Frog ". *Ibid.* p. 627. (Read June 18, 1857.)

[24] ALISON's *Outline of Pathology and Practice of Medicine*, 1843–4, p. 122.   "These facts afford a strong presumption that in all these cases the impressions made on the capillaries and on the blood contained in them, solicit the flow through them on the *principle of a vital attraction of the blood*, rather than of relaxation of the vessels."

[25] JOSEPH, BARON LISTER. *Collected Papers.* pp. 219–21, " Vital Affinities." *Ibid.*, pp. 250–1, " Paralysis of Concentrating and Diffusing Forces."

[26] *Proceedings of the Royal Society of London*, vol. ix, No. 32.

[27] *Lord Lister*, by SIR RICKMAN GODLEE, p. 77.

[28] *Lancet*, 1864, March 12, 19, 26.

EXTENSIVE LOSS OF TIBIAL DIAPHYSIS.
TIBIO-FIBULAR GRAFTING.

By G. R. GIRDLESTONE,

HON. SURGEON, WINGFIELD-MORRIS ORTHOPÆDIC HOSPITAL, AND HON. ORTHOPÆDIC SURGEON, RADCLIFFE INFIRMARY, OXFORD.

AND W. B. FOLEY,

ASSISTANT SURGEON, WINGFIELD-MORRIS ORTHOPÆDIC HOSPITAL, AND HON. ASSISTANT ORTHOPÆDIC SURGEON, RADCLIFFE INFIRMARY, OXFORD.

THE difficulty, or even impossibility in some cases, of bridging a big gap in the tibia after a partial diaphysectomy for osteomyelitis is well known to all who practise bone surgery, and to meet it a new method of bone-grafting was devised and carried out in the case to be described. When recourse is had to direct grafting from the upper to the lower tibial fragment it means that the graft must lie with several inches of its length in tissues which have been septic, are heavily cicatrized, and poor in vascularity, with the result that only too often the central part of the graft dissolves and disappears. Furthermore, the ends of the tibia are generally pointed. sclerosed, and frequently contain small residual foci which lead to infection of the wound and sequestration of the graft. To obviate these great drawbacks it was decided to make use of the fibula to provide a clean, osteogenetic, and vascular bed for the grafts destined to transmit body weight from upper to lower tibial metaphysis. The operation is applicable whenever part of the upper and lower tibial metaphysis remains ; and body weight is still transmitted, as it should be. from the upper tibial articular surface to the lower one, the only difference being that *between* these points the weight is transferred to the fibula. The central portion thus consists of fibula strongly reinforced by grafting, and at each end a flying-buttress graft transmits the weight from tibia to fibula above, and back from fibula to tibia again below. The details of operative technique together with the case notes. diagram, and radiographic appearances are appended.

A   B   C

FIG. 260.—A, Delagenière graft, i.e., periosteum with a thin leaf of bone under it taken from almost the whole breadth of subcutaneous surface of tibia. B and C, Albee grafts (cut from area already denuded by the cutting of A): B. Long. to reinforce the shaft of fibula : C, Divided into two grafts to be pegged obliquely from fibula into upper and lower metaphyses of tibia.

Operative Plan.—The idea. in broad outline, was to induce the shaft of the fibula to become as strong as that of the tibia—or at all events, to make it a strong solid bone capable of carrying on by itself the weight-bearing function of the limb. It was proposed to do this by embedding the

grafts obliquely from the fibular shaft into the healthy tibial metaphyses above and below; and to facilitate and accelerate the strengthening of the fibular shaft by reinforcing it with two long grafts, one of the Albee and one of the Delagenière type (*Fig.* 260), these grafts to be laid in a vascular or osteogenetic bed prepared by turning inwards and backwards an osteoperiosteal flap from the front and the inner side of the fibular shaft. It was expected that this strong reinforcement would give the fibular shaft a fair start in its task of making itself competent to carry the weight of the body. A study of the radiograms will show that these hopes have been realized.

CASE HISTORY.

C. C., at that time aged 3 years, was first seen in September, 1927. He was then in the wards of a local general hospital, where he had been for one year, since the onset of acute osteomyelitis of his left tibia in September, 1926. He had had three operations at the hospital, and had a still unhealed sinus over the shaft of his tibia, which was extremely and unduly mobile owing to loss of bone substance. His general health was satisfactory and he was fitted with a weight-bearing caliper and guarding plaster, and allowed to get up.

Four months later (January, 1928) he was admitted to the Wingfield Orthopædic Hospital with a metastatic abscess of the hand. X-rays at this time showed that all the central portion of the tibial shaft had disappeared, and that the ends of the upper and lower fragments were dense and ragged.

This abscess in the hand was opened and drained, and at the same time the tibial sinus was excised and granulation tissue removed from between the bone-ends. It was then left to heal by granulation after packing with vaseline and B.I.P.P.

Two months later (March, 1928) the patient was discharged, with the tibial wound healing well by granulation, in a caliper and plaster with window for dressings.

In June of the following year he was readmitted. The tibial

FIG. 261.—X-ray, May 24, 1930, showing the tapered ends with a skin of sclerosed bone, and an oval area in the upper fragment suggestive of a residual abscess.

sinus was still not quite healed, so a further operation was done consisting of excision of the sinus track down to and including about an inch of the upper end of the lower tibial fragment. The spiky lower end of the upper tibial fragment was also removed to prepare the way for grafting at a future date. At that time an ordinary tibial graft was contemplated.

A series of metastatic abscesses in various parts of the body now followed, and these were opened and drained, diseased bone and granulation tissue being removed in some cases. The patient was given a course of collosol manganese injections. These abscesses continued up till February, 1930. From this date till June, 1930, he was free from septic manifestations, and was allowed to be up and about in a caliper and plaster, though still an in-patient.

BONE-GRAFTING OPERATION (*Figs.* 262, 263).—This was performed by one of us (G. R. G.) on July 31, 1930. Through a long external incision practically the whole length of the shaft of the fibula was exposed and the periosteum incised and reflected. Tracks were then prepared through the soft parts deep to the anterior tibial muscles, vessels, and nerves, beginning from each end of the exposed fibular shaft and continued as tunnels drilled with a narrow gouge into
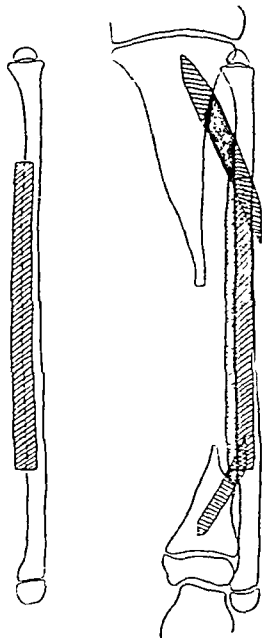
FIG. 262. FIG. 263.

FIG. 262.—Diagram illustrating the reflection of an osteoperiosteal flap from front and inner side of the shaft of fibula.

FIG. 263. — Tracing from X-ray showing the long vertical and oblique peg grafts. In addition (shown stippled) there is an indication of the placing of the Delagenière graft over the Albee (which lies sandwiched between this and the deeper-lying osteoperiosteal flap from the fibula).

FIG. 264.—X-ray, Oct. 2, 1930, two months after the grafting operation.

the healthiest and strongest part of the tibial metaphysis, the upper one obliquely upwards into the upper metaphysis, and the lower one obliquely downwards into the lower metaphysis.

The right tibia was then exposed and four grafts cut as follows : (1) An osteoperiosteal graft about ¾ in. broad and sufficiently long to cover the whole length of fibula exposed : (2) A graft about ⅜ in. broad, consisting of cortical and medullary bone, of the same length : (3) Two similar but shorter and rather stouter grafts. The two short grafts, after being pointed slightly at one extremity, were pegged firmly into the tunnels previously drilled in the upper and lower tibial metaphyses.

The free ends of these grafts, after traversing the oblique tracks in the soft parts, ended in contact with the bared shaft of the fibula under the reflected periosteum. The ends were crushed and turned so as to lie in very close apposition to the fibular shaft.

The long bone-graft was then laid along in contact with the shaft of the fibula under the fibular periosteum, and the osteoperiosteal graft was laid over it with its bony surface in contact with the fibular shaft and the three grafts. Both wounds were sutured, and the grafted leg put into a plaster case including knee- and ankle-joints.

Three months later (October, 1930) the plaster was changed. X rays at this time showed all the grafts 'taking' well, and an increase in thickness of the fibular shaft beginning to take place, especially on the anterior surface (*Fig.* 264).
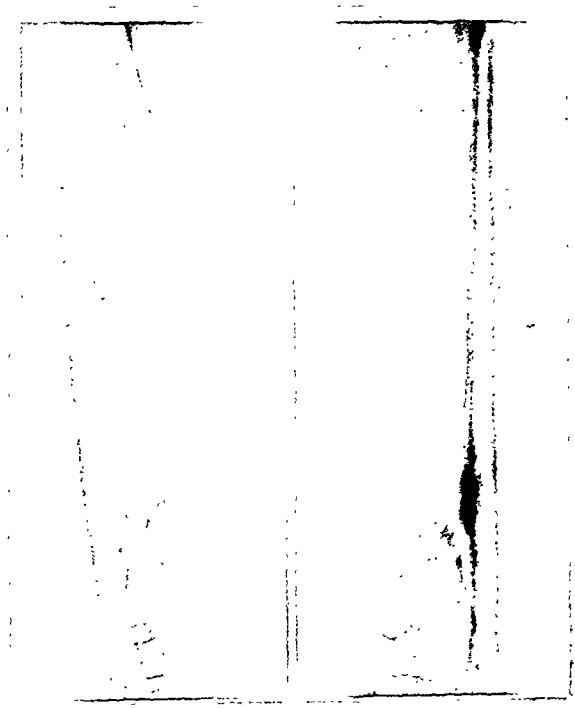
FIG. 265.—X-ray, Dec. 5. 1930, four months after the grafting operation.

A further X-ray (*Fig.* 265) taken two months later (December, 1930) shows a considerable further increase in the solidity of the grafts and resultant thickening of the fibular shaft.

Clinically the leg now felt firm and solid, having lost entirely the old feeling of undue mobility in every direction, due to the gap between the tibial ends. A light modified Delbet plaster was then applied and the patient allowed to walk on this.

The latest X-ray, taken in April, 1931 (*Fig.* 266), shows great hypertrophy of the fibular shaft, which is beginning to resemble in thickness the tibia of a child of this age. The boy is still walking with his Delbet plaster, but this will be removed shortly and walking without it allowed a year after the grafting operation. *Fig.* 267 shows the patient's condition on Nov. 4, 1931.

FIG. 266.—X-ray, April 2, 1931, eight months after the operation.

COMMENTS.

It is probable that in subsequent cases bone-grafting by this method would be resorted to even earlier than five months after subsidence of septic manifestation, local or general. Even this interval, however, is short compared to the time that would have to elapse before direct end-to-end grafting of the tibia, through the old inflammatory area, could even be considered.

(N.B.—23.11.32. There is now no shortening, and the boy, in his father's words, "runs about as well as anybody.")

FIG. 267.—Condition of patient Nov. 4, 1931.

A CASE OF PARATHYROID TUMOUR ASSOCIATED WITH GENERALIZED OSTEITIS FIBROSA.

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THE case we report below is further testimony to the view, so ably presented by Hunter in this country, that generalized osteitis fibrosa (von Recklinghausen) is a clinical manifestation of hyperparathyroidism.

CASE REPORT.

D. D., single woman, aged 48 years (L.R.I. 27270/1932).
COMPLAINTS.—Pain and swelling of jaw, head, and legs.
HISTORY.—
1923.—First noticed painful swelling of the right lower jaw, which rapidly increased in size for a few months and then remained stationary till twelve months ago, since when a further increase in size has been observed.

1928.—Appendicectomy and ? gastric ulcer removed.

1929.—Generalized headache with gradual increase in size of head.

1930.—First noticed swelling of upper end of right tibia.

July, 1931.—X-ray urinary tract : " Two large and at least two small stones in right kidney. Very suspicious of presence of small stones in left kidney. Abrodil shows hydronephrosis of right kidney."

August, 1931.—Right nephrotomy by Mr. W. R. Williams ; four stones removed.

FIG. 268.—Facial appearance and swelling of lower jaw.

Family History.—No bone disease. Father and mother died of 'senile decay' at 83 and 85 years respectively. Three brothers and four sisters well. One sister has phthisis.

ON EXAMINATION.—Weight 8 st. 10 lb. Height 5 ft. 4 in. Normal physical development.

Appearance and jaw swelling seen well in *Fig.* 268. Skull normal to palpation and auscultation, but tender on pressure ; upper end of right tibia

swollen and tender; no other bone deformity. No anæmia, no clubbing of fingers; sclerotics bluish. No tumour felt in neck.

Alimentary System.—Artificial teeth, tongue clean and moist, no tonsillar or pharyngeal infection, sinuses and antra transilluminate normally. Abdomen: generalized visceroptosis with palpable liver and spleen and right kidney; scars of previous operations well healed. Rectal examination negative.

Cardiovascular System.—Heart normal in size, shape, sounds. position, and rhythm, but systolic bruit audible at all areas, especially mitral. Blood-pressure, 135/70; pulse-rate, 84. No arteriosclerosis, either peripheral or retinal.

Central Nervous System.—No diplopia or visual defect. Fundi, pupils, reflexes, sensation, co-ordination, etc., reveal no evidence of organic nervous disease.

Respiratory System. — Lungs clear.

Fig. 269.—Antero-posterior view of skull.

Fig. 270.—Lateral view of skull.

Genito-urinary System. —No dysuria; no frequency. Menses irregular and scanty. Urine: acid, specific gravity 1008; trace of albumin; no Bence-Jones protein; no sugar; no excess of urobilin; few leucocytes and oxalate crystals in deposit.

Blood.—Hæmoglobin, 93 per cent; red blood-corpuscles, 4,900,000; white blood - corpuscles. 8240; normal differential count. Coagulation and bleeding time, normal. Wassermann reaction, negative.

RADIOGRAMS.—Dr. R. E. Roberts, May, 1932.

Pelvis.—There is no deformity and no increase in size or thickness of the pelvic bones.   In the right innominate bone are seen small areas of slightly exaggerated lamellation, together with small translucent areas suggestive of cyst formation.

Lumbar Spine. — The vertebral bodies show no alteration in shape and the intervertebral spaces are normal.   The normal texture of the bones has been altered, the upper and lower margins showing a homogeneous slight increase in density, whilst in the central portions are irregular areas of opacity and translucency without any suggestion of lamellation.

Skull (*Figs.* 269, 270).—The calvarium shows a slight thickening with loss of differentiation between the

FIG. 271.—Radiogram of mandible.

inner and outer tables and diploë.   Irregular areas of translucency are seen, some on the outer surface and some extending through the whole thickness of the bone.   The base of the skull shows no thickening.   The pituitary fossa

FIGS. 272, 273.—Radiograms of left and right tibiæ.

is normal in size, and the sphenoidal and frontal sinuses show normal appearances.

Mandible (Fig. 271).—The mandible on both sides shows a marked irregular thickening of both the horizontal and the ascending rami. This is more marked on the right side than on the left. The normal texture is lost and the whole bone presents a coarse opaque basis, throughout which are scattered numerous irregular translucent areas, some of which are clearly defined and suggest cysts. (Very little change has taken place since the previous X-ray examination of Oct. 10, 1930.)

Left Tibia (Fig. 272).—The anterior border of the upper two-thirds of the tibia shows a marked subperiosteal thickening which produces a convexity of this border. The medulla is encroached upon slightly by the cortical thickening. There is a slight tendency to the formation of irregular vertical striæ. The tuberosities and lower end of the tibia show normal X-ray appearances, as also does the fibula. Though the anterior tibial border is convex there is no actual bowing of the tibia.

Right Tibia (Fig. 273).— In the upper third of the tibia there is a cystic area with fairly well defined upper and lower edges. The cortex in this region is thinned anteriorly, but there is only a very slight expansion; the anterior surface of the bone bulges slightly. but merges with the surface of the bone lower down where the cortex shows slight thickening.

Urinary Tract.—Shadows in left renal area suggestive of numerous small stones and one suggestive of stone in left

Fig. 274.—Showing calcium content of serum and inorganic phosphorus of plasma before and after removal of parathyroid tumour.

ureter. Shadow behind symphysis pubis suggestive of small stone in bladder.

Calcium and Phosphorus Metabolism before Operation (*Fig. 274*).— Hypercalcæmia was constantly present before operation. varying between 13 and 17·4 mgrm. per 100 c.c. The plasma inorganic phosphorus was 2·4 to 2·6 mgrm. non-protein nitrogen was 33 mgrm., and uric acid was 4 mgrm. per 100 c.c. The average calcium excreted per three-day period on a diet containing 150 mgrm. calcium daily was 0·63 grm. in urine and 0·51 grm. in fæces.

OPERATION (June 6, 1932).—Although there was no apparent swelling in the neck during repose, a distinct fullness was noted in the right side of the neck when it was stretched backwards over the sand-bag before the incision was made. The anæsthetic used was intratracheal ether. After the usual collar incision the infrahyoid muscles were divided transversely, when the fullness previously noted was seen to be due to a swelling immediately below the right lobe of the thyroid gland.

This tumour (the right inferior parathyroid) was oval in shape and measured 2·5 × 1·5 × 1·0 cm. and was slightly lighter in tone than the thyroid gland itself, which it had displaced upwards. It was situated immediately below the right lobe of the thyroid, which covered its upper third. The large mass of tumour lay between the trachea and the carotid sheath. It was completely exposed by turning inwards the right lobe of the thyroid, to which it was attached by a few delicate adhesions ; after dissection of these the main blood-vessel supplying the tumour (a small branch of the inferior thyroid) was ligated and the tumour removed.

FIG. 275.—Right inferior parathyroid.

A further search was made for other parathyroid tumours. There was none on the right side, but on turning the left lobe inwards a small tumour was found in the upper part of the left lateral lobe. This was completely buried in the substance of the thyroid and was removed. It proved to be a thyroid adenoma. A portion of bone was removed from the right tibia.

PATHOLOGICAL REPORT (Dr. H. H. Gleave).—

1. *Right Inferior Parathyroid* (*Fig.* 275).—This is a firm pale fleshy tumour measuring 2·5 × 1·5 × 1·0 cm., weight 2·5 grm.

Microscopic (*Fig.* 276).— This shows an enlarged parathyroid, the enlargement consisting of an increase in the principal cells. These are highly vesiculated. Some of the nuclei are greatly enlarged, and a few cells contain several nuclei. Dark oxyphil cells are scanty. Some of the acini have a central lumen containing acidophilic material.

2. *Small Tumour from Upper Pole of Left Lateral Lobe of Thyroid* (*Fig.* 277).— The tumour measures 1 cm. in diameter. It shows large thyroid vesicles containing thin colloid and lined by flattened cells.
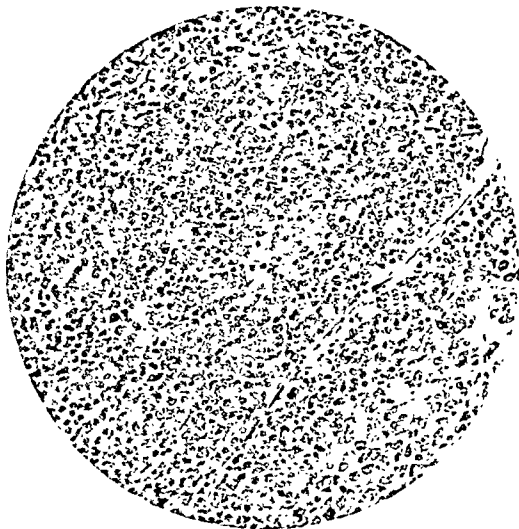
FIG. 276.—Microscopic appearances of parathyroid. (× 140.)

3. *Piece of Right Tibia.*—Specimen is an irregular piece about 1 cm. cube.

Macroscopic.—It has a granular appearance, and cuts easily, but requires decalcification for preparation of sections.

Microscopic.—The structure is almost uniform (*Fig.* 278). Numerous bone trabeculæ, measuring about 50 to 100 μ in width. and in length up to 0·5 mm., are seen lying in a rather greater amount of fibrous tissue. The trabeculæ are frequently surrounded by a narrow zone of osteoid tissue. Osteoblasts. applied to the trabeculæ. are numerous. Osteoclasts are few. They lie both in the fibrous tissue and applied to the trabeculæ. They have very numerous nuclei which fill the cell, and a smaller amount of cytoplasm than usual. The fibrous tissue between the trabeculæ consists of very numerous spindle cells and delicate collagen fibrils. In it are numerous wide capillaries with a wall of a single layer of endothelium, and a few small arterioles.
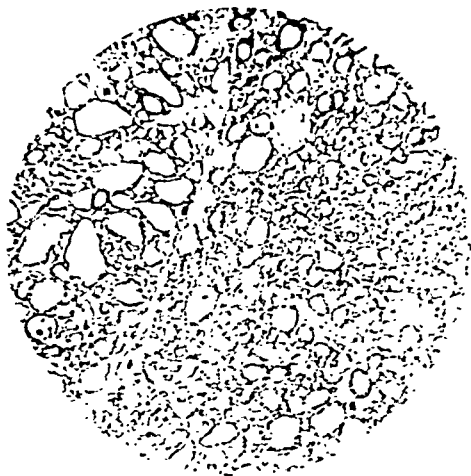
Fig. 277.—Thyroid adenoma. (× 50.)

The condition is thus *osteitis fibrosa.* Formation of imperfect bone is in excess of resorption.

POST-OPERATIVE PROGRESS.—

June 6, 1932. — 10 p.m. (eight hours after operation): Comfortable. no evidence of latent tetany.

June 7.—9.30 a.m.: Slight tingling in fingers and toes; no latent tetany demonstrable by muscle irritability. 6 p.m. : No pain or tenderness of affected bones.

June 8.—10 a.m. : Blood calcium 8·7 mgrm. per 100 c.c. No tetany.

Fig. 278.—Bone from right tibia. The dark masses are nuclei of osteoclasts.

June 9. — 8 a.m. : Well-marked Chvostek sign, and twitching of left face. No Trousseau's sign. Intravenous calcium gluconate (10 c.c. of 10 per cent solution) and 20 units of parathormone given with relief of tingling and facial twitching. These injections were repeated on the three following days.

June 14.—No recurrence of tetany. Commenced high calcium diet and calcium lactate 20 gr. t.d.s.

CALCIUM AND PHOSPHORUS METABOLISM AFTER OPERATION.—The serum calcium dropped to 8 mgrm. per 100 c.c. after operation (*see Fig.* 274) and the plasma inorganic phosphorus was 2·8 mgrm. A month after operation the average calcium excreted per three-day period on a diet containing 150 mgrm. calcium daily was 0·2 grm. in the urine and 0·45 grm. in fæces.

COMMENT.

The bone changes, the parathyroid tumour, and the pre- and post-operative changes in calcium metabolism are typical. The patient presented, however, one feature which calls for comment. On purely radiological evidence she had previously been regarded as possibly suffering from leontiasis ossea, osteitis deformans, chronic osteomyelitis, etc. This emphasizes again the necessity for full metabolic investigation in cases of generalized bone disease.

The metabolic investigations undertaken in our case proved the true nature of the underlying condition, and, in the light of recorded experience, the presence of a parathyroid tumour was confidently predicted.

ADDENDUM.

Two recent radiographs of the skull and tibia (*Figs.* 279, 280) which are comparable to *Figs.* 270 and 273. Note the increased density of the upper end of the tibia and the increased calcification in the skull with greater clarity of outline, particularly in the vascular markings. (Sept. 19, 1932.)

FIG. 279.

FIG. 280.

THE DIAGNOSIS AND TREATMENT OF GENERALIZED OSTEITIS FIBROSA WITH HYPERPARATHYROIDISM.

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A NUMBER of recent publications have shown clearly that there are cases of generalized osteitis fibrosa associated with hyperactivity and tumours of the parathyroid glands, and that in these cases benefit is derived from the extirpation of the parathyroid tumour or tumours. The relevant literature has been reviewed by Barr and Bulger (1930), and in this JOURNAL by Hunter and Turnbull (1931), and will not be referred to in this paper except in particular instances. If the diagnosis of generalized osteitis fibrosa and hyperparathyroidism is made, an operation to explore the parathyroid glands is not only justifiable but is indicated. In recording three further cases of this association of generalized osteitis fibrosa with hyperparathyroidism which have been successfully treated by removal of parathyroid tumours, we wish to stress the diagnostic criteria and the difficulties that may arise in the operative treatment. With this object (1) the histories of the three cases, (2) the bony lesions as determined from clinical and X-ray examinations, which in two of the cases covered periods of six and seven years, (3) the results of the chemical investigations, and (4) the operative treatment and the tumours removed, are described and discussed. Brief reference will be made to a fourth case that was studied in the same manner, and in which no parathyroid lesion was found at operation, because the difference between the findings in this case and in the three in which parathyroid tumours were found, point to some of the essentials for diagnosis.

CASE HISTORIES.

Case 1.*—The patient was a married woman, aged 42 years, engaged in housework and in general work on her husband's fruit farm. Her father died of phthisis ; her mother, two sisters, and her husband were in good health. She had measles in infancy, jaundice at the age of 12, and tonsillectomy was performed when she was 22 years of age. She had attacks of severe frontal headaches and nausea all her adult life, at monthly intervals and lasting two days. Dysmenorrhœa had continued since puberty ; she had never been pregnant.

In 1920, at the age of 32, pains in the left arm occurred after exertion. In 1921 a fracture of the right humerus above the elbow occurred, and healed quickly. In 1922 there were pains in the shoulders, attributed to neuritis. In 1924 a spontaneous fracture occurred in the right humerus, and she was admitted to the

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* The earlier clinical history of this case and reproductions of X-ray photographs of some of the bones were published by one of us (R. C. E.) in the *Robert Jones Birthday Volume* (1928).

Royal National Orthopædic Hospital and kept under observation there after leaving the hospital. Cyst-like expansions were discovered on X-ray examination in several of the long bones. Two of the cysts were curetted. During the following years further cysts appeared. Most of these were curetted as they expanded and seemed likely to lead to fractures. From 1927 onwards, progressive rarefaction of the tibiæ was noted, while the skull became thickened. In 1925 the serum calcium was 9·8 mgrm. per 100 c.c. ; in 1927 the serum calcium was 14·6 mgrm. and 14·8 mgrm. per 100 c.c. on two occasions, and the blood phosphorus was 1·6 mgrm. and 1·1 mgrm. per 100 c.c.

She was admitted to St. Bartholomew's Hospital in January, 1930, and was then a small alert woman, looking older than her years, with pains in the limbs, and considerably limited in her activities. The muscles of the limbs were wasted. The skull was not large, but the forehead was prominent and showed definite bossing. The bones of the face were symmetrical, and nothing abnormal was noted in the vertebral column and ribs. The arms and legs showed numerous scars of operations, and the left arm was in plaster because of a recent fracture of the humerus. The long bones had many irregularities in their outlines and there was marked bowing of the humeri and tibiæ. The fingers and toes appeared normal. The alimentary, respiratory, cardiovascular, and nervous systems appeared normal, but the blood-pressure was—systolic 150 mm. of Hg, and diastolic 105. The urine was normal, and X-ray examination of the kidneys failed to show the presence of renal calculi. In the neck the right lobe of the thyroid appeared larger than the left. The results of the operations on the bone cysts and of the X-ray examinations of the bones, and the results of the chemical investigations, are given in later sections.

An operation on the neck was performed on May 8, 1930, and a parathyroid tumour removed from the left side. Symptoms and signs of tetany appeared during the week following the operation, and gradually increased in severity. After four doses of Collip's parathyroid extract, 5 units twice a day, the tetany disappeared and did not recur. Following the operation the patient rapidly grew stronger, and a month later returned home. Six months after the operation she reported that she was suffering from menorrhagia, and still had some pains in the limbs, but no further fractures had occurred. No increase in the density of the bones could be demonstrated, but she was carrying on her normal occupation with greater comfort and efficiency.

She was admitted for a few days in May, 1932, two years after the removal of the parathyroid tumour. She was in good health, free from pain or other symptoms, and working hard on her farm. There was no striking change on physical examination, except that the blood-pressure had risen to systolic 220 mm. of Hg, diastolic 130. There was no increase in body weight. X-ray examination of the bones showed that definite improvement had occurred in them, and these changes are described in a later section. The serum calcium was 11·0 mgrm. per 100 c.c., and the serum phosphorus 3·2 mgrm., which are normal values. She states that she is frequently subjected to traumata that would previously have caused fractures but now have no effect.

Case 2.—The patient was an unmarried woman of 26 years, who worked as a clerk in the Civil Service. Her family were healthy, and she had had no serious illnesses, but had never been robust.

In December, 1928, an attack of influenza was accompanied by aching of the bones, which persisted and grew steadily worse, was increased by exercise and improved with rest. In May, 1929, a tumour, described as a fibrous epulis, was removed from her jaw. In January, 1930, wasting of the muscles of the shoulder and pelvic girdles was noted and considered to be due to a myopathy. In July, 1930, a fracture of the head and neck of the femur followed a slight injury, and she was admitted to St. Bartholomew's Hospital.

She was a thin nervous woman, free from pain when at rest in bed. The alimentary, respiratory, cardiovascular, and nervous systems appeared normal. A bacilluria due to *B. coli* was present, and X-ray examination of the kidneys some months later showed calculi in the pelves of both kidneys. The left humerus was

expanded in the upper third, and the outline of the right tibia was irregular; the fracture of the left femur appeared to be healing satisfactorily. A small localized swelling was seen and felt near the lower pole of the right lobe of the thyroid gland. The results of the X-ray examination of the bones and of the chemical investigations are described in later sections.

An operation on the neck was performed on Sept. 15, 1930, and a parathyroid tumour was removed from the right side. The recovery was uneventful. A month after the operation severe pain developed in the right leg, but no evidence of fracture was obtained, and the pain gradually subsided. Pains in the bones persisted for some months but gradually lessened, and six months after the operation they were only present on considerable exertion. Menstrual loss was increased, but the patient was gaining weight and strength. In June, 1931, she had so improved in general health and in strength that she applied for reinstatement in her post in the Civil Service. X-ray examination showed definite increase in the density of the bones.

In May, 1932, twenty months after the removal of the parathyroid tumour, she felt well and appeared healthy. She had regained her muscular strength and had put on nearly a stone in weight. A few days previously she had slipped in the street and fallen without any fracture resulting. X-ray examination of the bones showed striking improvement. The changes in the bones following the operation are described in a later section.

Case 3.*—The patient was an unmarried girl, aged 23 years. Her parents and nine brothers and sisters were alive and healthy, and she had been free from serious illness in infancy and childhood.

When 15 years old, a pain developed in the left leg that caused her to limp. She was admitted to a hospital, and extension applied. Following this she was admitted to five different hospitals on several occasions, where the treatment adopted in most instances was the putting of the left leg in plaster; the left femur had sustained a spontaneous fracture at the age of 16. At this time the right leg became painful and a spontaneous fracture occurred in the right femur, and when she was 17 years old, swellings appeared in the fingers, wrists, and sternum. When 18 years old, the upper part of the left tibia was much enlarged and a pulsating swelling over its inner aspect was diagnosed as an osteoclastoma. Later the back became bent, and a swelling appeared in the left humerus, but the enlargement of the head of the left tibia had ceased to pulsate. When 22 years old, the left thumb and left femur were curetted at the Royal National Orthopædic Hospital, and a year later a spontaneous fracture occurred in the left humerus. During most of her illness she had been bedridden. She complained of few symptoms apart from the bones; the digestion was good; there were no symptoms of urinary trouble; menstruation had been normal, but became irregular and then ceased while under our observation in hospital.

She was admitted in September, 1930. The skeleton was grossly deformed, and there was much muscular wasting. The skull was irregularly enlarged, the face appearing small below it. The spine was twisted and shortened. The thorax showed a long antero-posterior diameter, the sternum was prominent and its surface irregular, and the lower ribs splayed out. The long bones were bowed, and had irregular expansions at the extremities and along the shafts. The fingers were similarly irregular and thickened. She was bright, intelligent, and co-operative. No disease of the nervous system, heart, or lungs was discovered. The lower pole of the left kidney was palpable; the urine contained albumin and many pus-cells, and X-ray examination showed calculi in the pelves and calices of both kidneys. Urea clearance tests gave a standard clearance of 18·5 c.c. and a maximal clearance of 34·5 c.c. of blood per minute, as compared with the normal figures of 54 c.c. and 75 c.c. The basal metabolic rate was −22 per cent. Blood-count : red blood-cells,

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5,100,000 ; white blood-cells, 9,500 ; hæmoglobin, 92 per cent. The results of the operations on the bones and of the X-ray examinations of the bones, and the results of the chemical investigations, are described in later sections.

On Jan. 19, 1931, an operation was performed on the neck and a parathyroid tumour removed. Two days after the operation symptoms of tetany appeared. Injections of Collip's parathyroid extract were given for the following five days in doses of 10 units daily, when the symptoms of tetany disappeared, and calcium lactate, by the mouth, and radiostoleum were given for a further period of some weeks. Sixteen days after the operation the patient was discharged from hospital, but returned five months later for further observation. During this interval she gained considerably in strength and felt better. There was no change in outward appearance ; X-ray examination of the bones and kidneys showed no change ; pus was still present in the urine ; and the urea clearance tests showed no improvement in renal function.

She was seen again in May, 1932, sixteen months after the removal of the para-thyroid tumour. She felt well and was leading an active life at home. She seemed happy and had gained nearly a stone in weight. There was little change in the appearance of deformity, but she was able to do without crutches except when walking in the street. Menstruation was normal once more. X-ray examination of the bones showed definite changes that are described in a later section.

EXAMINATION OF THE BONES AND CYSTS.

Case 1.—When this patient was admitted to the *Royal National Orthopædic Hospital* in December, 1924, there was a flail-like condition of the shaft of the right humerus just above the elbow. X-ray examination demonstrated cystic expansions in the right and left humeri, left radius, left femur, and right tibia. In the right humerus the bone was completely absent for a space of about two inches in the lower part of the shaft, and there was a second large cavity showing only a vague bony outline occupying the whole of the upper part of the shaft. The bone between these two cavities was much rarefied.

A cavity in the right tibia was explored for diagnosis, and a simple cyst containing brown fluid found. There were no solid contents and the wall consisted of smooth bone. Three weeks later the two large cysts in the right humerus were opened. In each case the cavity was covered by fibrous peri-osteum, and it was bounded above and below by the irregular bone of the humerus. The cavities were curetted, so that after the operation the humerus consisted of three portions connected by two periosteal tubes. The arm was supported by a leather case, and the patient returned home. Three months after the operation the bone had consolidated ; the arm was out of shape, but had become quite strong and useful. The cysts, however, did not disappear completely ; the upper one refractured later and both cysts had enlarged again to a considerable size on X-ray examination in 1930.

From December, 1924, onwards the patient was kept under observation, the cysts in the various bones were watched, and whenever one appeared large enough to suggest the risk of a fracture it was opened and curetted. In every case the cyst thus curetted filled up to some extent and the bone became stronger, but as a rule the cystic condition remained evident on X-ray examination. A large cyst in the upper part of the shaft of the right radius was aspirated without being opened. This did not cause consolidation of

the cyst, which eventually expanded and became very painful. It was finally opened, and after this it filled up and the bone became stronger.

The cysts explored had no solid contents, and with one exception no proper lining. One cyst in the middle of the shaft of the left femur, which had caused severe pain, was lined by a thick grey membrane, which in places showed fibrous tissue and small fragments of bone, but which also showed quite definite areas of organizing blood-clot. The appearances suggested that there had been a recent hæmorrhage into this cyst, and that the lining membrane had been formed by organization of the clot.

Histological investigation of bone curetted from the margins of the cysts showed in one case that the bone was being absorbed by osteoclasts. Except for this, nothing was found to explain the way in which the cysts were formed, and in no case was there any evidence of decalcification of the bone at the margins of the cysts. No giant-cell tumours were found, nor was there any evidence of fibrosis of the marrow, which appeared normal in the neighbourhood of the cysts. There was nothing in the X-ray photographs to suggest the massive formation of fibrous tissue in the interior of the bones such as is seen in other forms of cystic disease of bone.

During the seven years 1924–30 further cystic changes were found in the right and left humeri, the left radius, the right and left femora, the pelvis, and the right tibia. In the skull the calvarium was found to be much thickened, the maximum being

A   B

Fig. 281.—*Case* 1. Right radius and ulna (A) before, and (B) two years after, removal of the parathyroid tumour, showing increase in density of bone and increase in size of cyst in lower end of radius.

about $\frac{5}{8}$ in. as estimated by an X-ray photograph, and there appeared to be a cavity behind the frontal sinus which was possibly a small cyst. The texture of the bone of the calvarium was obviously abnormal, the X-ray shadow showing a mottled woolly appearance.

X-ray examinations of the shafts of the long bones away from the cysts showed from the first a certain amount of porosis with widening of the

Haversian spaces, and this appearance became much more marked by 1930. There was not thickening of any bone except the calvarium and no bending of the long bones except at the site of the cysts where fractures or crumpling occurred.

Re-examination of the bones by means of X rays, six months after the removal of the parathyroid tumour, showed that the cysts had not altered and that the cystic areas in the right humerus were still very large. Control skiagrams showed that there was still much porosis of the bones, and that there had been no improvement in the bones as seen by X-ray photographs.

In May, 1932, two years after removal of the parathyroid tumour, X-ray examination showed that the bones had improved generally in density and that the cortical parts were thicker. The old cysts in the right and left humeri had filled up to a considerable extent and become much more dense, but many of the cysts were still present, and one in the lower end of the right radius was actually larger than it was before operation, although the shafts of the radius and ulna were much better formed (*Fig.* 281).

Case 2.—On admission to St. Bartholomew's Hospital in July, 1930, X-ray examination showed the following abnormalities of the bones :—

FIG. 282.—*Case 2.* Skiagram showing dense opaque area at site of cyst in left humerus twenty months after removal of parathyroid tumour.

1. Pelvis—extensive areas with formation of cystic spaces.

2. Left femur—head rarefied, a large cyst in the neck through which the bone was fractured, and cystic patches involving the cortical part of the bone in the upper part of the shaft.

3. Right femur—cystic patches in the cortical part of the bone extending through the shaft so that there was practically no normal cortex.

4. Right humerus—cortex rarefied, Haversian canals showing plainly, and cystic cavities involving portions of the cortex.

5. Skull—calvarium thickened to about half an inch and showing mottled appearance.

Following the removal of the parathyroid tumour in September, 1930, further X-ray examinations showed a large cavity in the right femur a little below the small trochanter, and, when compared with controls, the bones of the right forearm showed much rarefaction with loss of definite cortex and extensive cystic patches, one of which in the radius caused expansion of the bone.

The patient was re-admitted in June, 1931, nine months after the removal of the parathyroid tumour, when she had improved considerably in health

and strength, and X-ray examinations showed the following changes, and indicated great improvement in the bony condition :—

1. Right femur—the large cystic area in the upper part of the shaft still clearly visible, but covered by a definite cortical layer, and the lower part of the shaft showing much thickening of the surface of the bone.
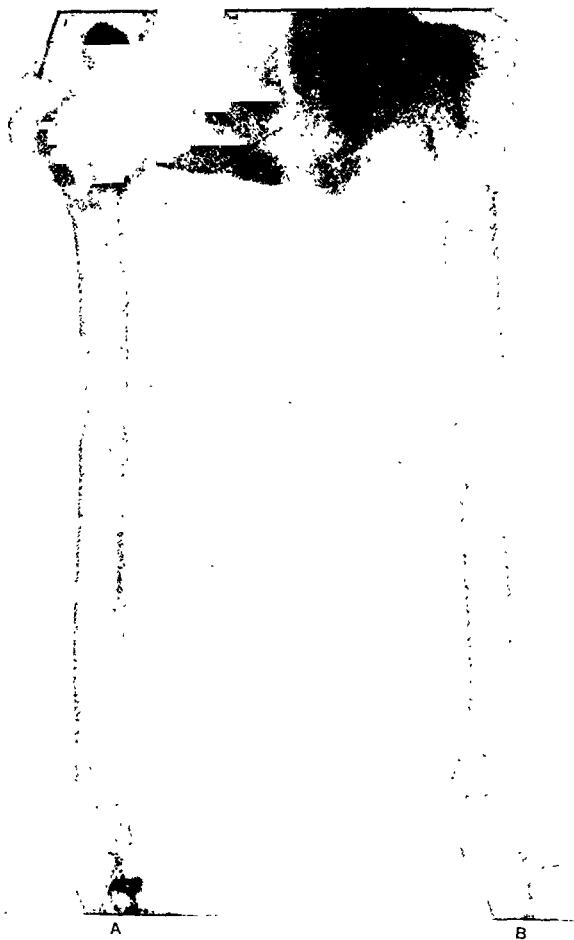
Fig. 283.—*Case 2.* Right humerus (A, before, and (B) twenty months after, removal of parathyroid tumour, showing disappearance of cystic cavities and increase in cortex and in density of bone generally.

2. Left femur—cystic areas no longer seen, cortex re-formed, but not dense, mottled in appearance, with irregular projections on the surface, and general increase in density of bone.

3. Right humerus—cystic areas no longer seen.

4. Left humerus—oval opacity below surgical neck, and oval expansion without opacity in lower part of shaft.

Fig. 284.—*Case* 2. Skull before removal of parathyroid tumour.

Fig. 285.—*Case* 2. Skull twenty months after removal of parathyroid tumour, showing diminution of mottling, thinning of bone, and greater distinctness of vascular channels.

5. Right tibia and fibula—extensive thickening and opacity of shaft in middle third, the fibula more than twice normal thickness.

In May, 1932, twenty months after the removal of the parathyroid tumour, X-ray examination showed that the cysts were all transformed into dense opaque areas, and the bones well formed and of good density (*Figs.* 282, 283). The right femur, left radius, and right tibia and fibula showed much new periosteal bone. The skull was less mottled and thinner: the outline of all the vascular channels had become distinct (*Figs.* 284, 285).

Case 3.—When this patient developed a limp at the age of 15, X-ray examination showed slipping of the upper epiphysis of the left femur. At the age of 16, when the first spontaneous fractures occurred, skiagrams made at St. Thomas's Hospital showed the following abnormalities :—

1. Tibiæ—very thin in texture and translucent, the right straight, the left a little collapsed at the upper end.

2. Radii—texture fairly good but Haversian spaces more evident than normally, lower ends much enlarged in region of epiphysial line and obviously porous in texture.

3. Ulnæ—texture fairly good but Haversian spaces more evident than normally, epiphysial cartilages widened but without the cupping of the diaphysis which occurs in rickets.

4. Hands—spiky periosteal projections from the surface of the phalanges. small cystic area in base of first phalanx of right thumb.

5. Spine—slight scoliosis, small renal calculi noted.

At the age of 18, X-ray examination at the Pyrford Hospital showed the following further changes :—

1. Left tibia—markedly collapsed in upper part.

2. Femora—very light in texture without any proper cortex, shafts collapsed and deformed.

3. Pelvis—triradiate.

4. Right humerus—upper end collapsed and deformed.

5. Radii and ulnæ—porosis, particularly at lower ends, where epiphysial cartilages were widened.

6. Hands—cyst in first phalanx of right thumb larger, cyst in metacarpal bone of left middle finger, periosteal new bone less marked.

7. Skull—calvarium greatly increased in thickness, appearing mottled and woolly.

At the age of 20 years, the head of the left tibia and the base of the first phalanx of the left thumb were explored and were each found to contain solid tissue with the structure of an osteoclastoma and a spindle-celled stroma containing a few islets of bone. The bone at the margin showed definite lacunar absorption by giant cells.

On admission to St. Bartholomew's Hospital at the age of 23 years in September, 1930. the condition of the bones was much the same as that already described, allowing for a little increase in the deformities.

On re-admission, three months after the removal of the parathyroid tumour, at a time when she had gained in strength and felt better, there was no change in the appearance of the bones on X-ray examination.

FIG. 286.—*Case* 3.   Right femur and part of pelvis (A) before, and (B) sixteen months after, removal of parathyroid tumour, showing increased density of bone surrounding the cystic cavities.
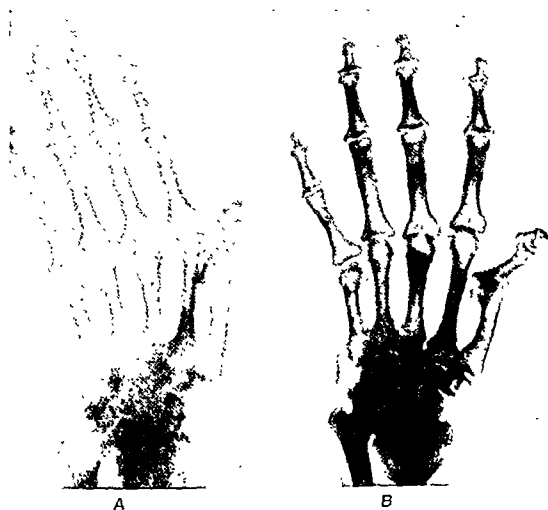
FIG. 287.—*Case* 3.   Left hand (A) before, and (B) sixteen months after, removal of parathyroid tumour, showing increase in density of bones generally and opaque area at site of cyst in third metacarpal bone.

In May, 1932, sixteen months after the removal of the parathyroid tumour, X-ray examination showed that the large cystic cavities in both femora still existed but were surrounded by denser bone (*Fig.* 286). The upper ends of the tibiæ had thickened and consolidated and consisted of rather irregularly dispersed cancellous bone. The cortex of the shaft of these bones was also thicker. A cyst in the third left metacarpal bone had been transformed into an opaque area (*Fig.* 287).

CHEMICAL INVESTIGATIONS.

Methods.—During the periods of study the diets of the patients were kept constant, and the calcium and phosphorus contents of the diets were calculated from the tables of Sherman (1924), confirmatory analyses being made in many instances. In most periods the quantities were those present in the patient's ordinary diet, about 1 grm. of calcium and 1·25 grm. of phosphorus a day, so that the relation of intake to output under conditions similar to those of their home life could be estimated. During certain of the periods diets low in calcium, containing 0·25 grm. or less of calcium, were given in order to make a comparison with the observations of other workers. The diets selected were given over a period of four or five days before any collection of excreta was made, in order that conditions should have time to become stabilized. The fæces were collected in four-day periods marked off by carmine. The urine was collected in two-day periods. The acid-base ratio of the diets was kept constant.

The calcium in the serum was estimated by Clark and Collip's (1925) modification of the method of Kramer and Tisdall (1921, 1923), the inorganic phosphorus in the serum by the method of Fiske and Subarrow (1925). The stools were ashed, following the method of Stolte (1911), the ash dissolved in dilute hydrochloric acid with the aid of heat, and the solution used for the determination of calcium and phosphorus. The urine was evaporated and ashed for the estimation of calcium ; the inorganic phosphorus was estimated directly in the forty-eight-hour specimens. In *Case* 3 the organic phosphorus in the urine was also measured. This proved to be so small an addition, that the omission of this measurement in the other cases cannot materially affect the results.

Results.—The results of the estimations are given in *Tables I, II, III*, and shown graphically in *Figs.* 288–290. In *Fig.* 291, the calcium and phosphorus balances of each of the three patients are averaged for the periods before and for the periods after the removal of the parathyroid tumour. In the same figure are included a normal balance recorded by Sherman (1924), and the balance in a case of generalized osteitis fibrosa with hyperparathyroidism recorded by Bulger, Dixon, and Barr (1930).

The results of the estimations before operation can be stated briefly as follows :—

The serum calcium (*Table I*) was abnormally high in two (*Cases* 1 and 2) out of the three patients. In *Case* 3 the serum calcium did not rise much above what may be considered a high normal value during the time the patient was under controlled observation, but an abnormally high value had

Table I.—Results of Calcium and Phosphorus Estimations.

CASE I.

Date	Diet — Grm. daily		Serum — Mgrm. per cent	
	Calcium	Phosphorus	Calcium	Phosphorus
6/2/30	Normal adult		14·9	2·0
13/2/30	1·040	1·259	14·7	2·2
20/2/30	1·040	1·259	16·2	2·6
10/3/30	0·250	0·800	15·8	2·0
17/3/30	0·250	0·800	15·6	2·8
8/5/30	Operation		—	—
26/5/30	0·986	1·053	5·9	2·9
2/6/30	0·986	1·053	5·4	4·0
15/12/30	Normal adult		8·2	—
—			—	—
—			—	—

CASE II.

Date	Diet — Grm. daily		Serum — Mgrm. per cent	
	Calcium	Phosphorus	Calcium	Phosphorus
23/7/30	Normal adult		16·4*	—
7/8/30	Normal adult		17·7*	—
14/8/30	1·022	1·262	15·6	2·5
21/8/30	1·022	1·262	15·5	2·2
15/9/30	Operation		—	—
16/9/30	Uncontrolled		12·4*	—
18/9/30	Uncontrolled		11·2	1·9
23/9/30	Normal adult		7·5	2·3
29/9/30	Normal adult		7·0	3·0
16/10/30	1·030	1·297	8·8	2·7
30/6/31	Normal adult		10·4	3·6

CASE III.

Date	Diet — Grm. daily		Serum — Mgrm. per cent	
	Calcium	Phosphorus	Calcium	Phosphorus
13/10/30	Normal adult		15·2*	—
27/10/30	0·753	1·092	12·7	2·1
14/11/30	0·099	0·572	12·1	1·35
17/1/31	Normal adult		11·8	2·6
19/1/31	Operation		—	—
21/1/31	Normal adult		7·3	2·3
21/4/31	0·753	1·092	9·1	4·3
11/5/31	0·099	0·528	9·3	4·1
—			—	—
—			—	—
—			—	—

* Observations made by others than the present writers.

been noted previously. The amount of calcium taken with the food had no effect on the level of serum calcium in these cases.

The serum phosphorus was abnormally low in all three cases.

On a high calcium diet (*Table II* and *Fig.* 291) the patients stored calcium in *Cases* 1 and 3, but in *Case* 2 there was a negative balance, the
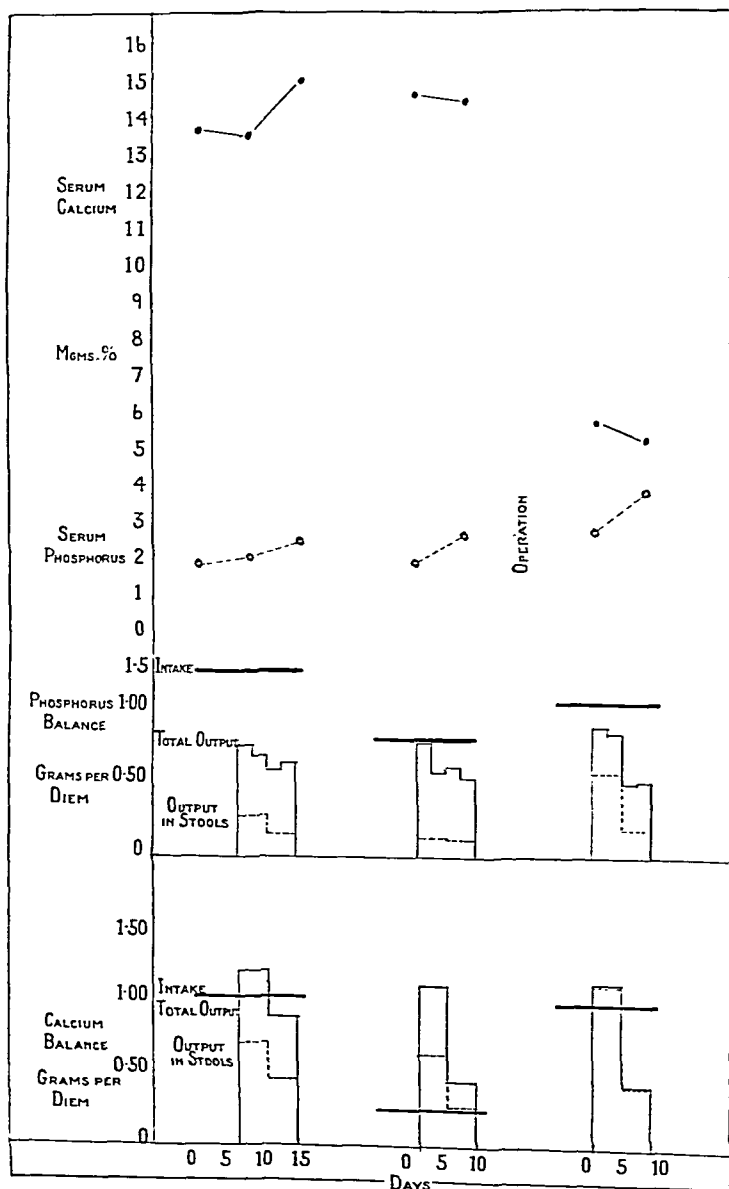
Fig. 288.—*Case* 1. Graphic representation of the calcium and phosphorus metabolism over two periods of eight days before parathyroidectomy and one eight-day period after.

Table II.—High Calcium Intake.

CASE	4-DAY PERIOD	SERUM		CALCIUM					PHOSPHORUS				
		Calcium (Mgrm. Per cent)	Phosphorus (Mgrm. Per cent)	Intake Daily (Grm.)	Faeces (Grm.)	Urine (Grm.)	Total (Grm.)	Balance	Intake Daily (Grm.)	Faeces (Grm.)	Urine (Grm.)	Total (Grm.)	Balance
I		14·9	2·0	—	—	—	—	—	—	—	—	—	—
	1	14·7	2·2	1·040	0·709	0·454	1·163	-0·123	1·259	0·272	0·476	0·748	+0·511
	2	16·2	2·6	1·040	0·462	0·430	0·892	+0·148	1·259	0·130	0·465	0·615	+0·644
		Operation											
	3	5·9	2·9	0·986	1·121	0·017	1·138	-0·152	1·053	0·570	0·286	0·856	+0·197
	4	5·4	4·0	0·986	0·425	0·010	0·435	+0·551	1·053	0·190	0·315	0·505	+0·548
II	1	15·6	2·5	1·022	0·446	0·483	0·929	+0·093	1·262	0·150	0·972	1·122	+0·140
	2	15·5	2·2	1·022	1·012	0·465	1·477	-0·455	1·262	0·404	1·039	1·443	-0·181
		Operation	7·0	3·0	—	—	—	—	—	—	—	—	—
	3	—	2·7	1·030	0·319	0·036	0·355	+0·675	1·297	0·154	0·346	0·500	+0·797
	4	8·8		1·030	0·542	0·028	0·570	+0·460	1·297	0·320	0·478	0·798	+0·499
III	1	12·7	2·1	0·753	0·752	0·159	0·906	-0·153	1·092	0·350	0·322	0·672	+0·420
	2	12·1	2·3	0·753	0·374	0·164	0·538	+0·215	1·092	0·157	0·157	0·614	+0·478
	3	—	—	0·753	0·374	0·160	0·534	+0·219	1·092	0·157	0·480	0·637	+0·455
	4	12·1	1·1	0·753	0·326	0·221	0·547	0·206	1·092	0·143	0·456	0·799	0·268
		Operation											

Table III.—Low Calcium Intake.

Case	1-Day Period	Serum Calcium	Serum Phosphorus	Calcium Intake Daily	Calcium Output Daily Faeces	Calcium Output Daily Urine	Calcium Output Daily Total	Calcium Balance	Phosphorus Intake Daily	Phosphorus Output Daily Faeces	Phosphorus Output Daily Urine	Phosphorus Output Daily Total	Phosphorus Balance
		Mgrm. Per cent	Mgrm. Per cent	Grm.	Grm.	Grm.	Grm.		Grm.	Grm.	Grm.	Grm.	
1	a	15·8	2·0	0·250	0·483	0·631	1·114	−0·864	0·800	0·143	0·515	0·658	+0·142
	b	15·6	2·8	0·250	0·262	0·187	0·449	−0·199	0·800	0·133	0·438	0·571	+0·229
III	a	12·1	1·4	0·099	0·099	0·148	0·247	−0·148	0·528	0·118	0·454	0·572	−0·044
	b	—	—	0·099	0·114	0·143	0·257	−0·158	0·528	0·114	0·362	0·476	+0·052
	Operation	9·1	4·3	—	—	—	—	—	—	—	—	—	—
	c	—	—	0·039	0·036	0·017	0·053	+0·046	0·528	0·055	0·344	0·399	+0·129
	d	9·3	4·1	0·090	0·051	0·015	0·066	+0·033	0·528	0·115	0·300	0·475	+0·053

patient excreting more calcium than she was receiving. On low calcium diets (*Table III*) negative balances were present in *Cases* 1 and 3. These diets cannot be considered adequate in calcium intake, and provided a more severe test of the ability to conserve calcium. When, however, the calcium excretions in the urine and in the fæces are considered separately, it is seen that in *Cases* 1 and 2 the urinary excretions are abnormally high on both high and low intakes, since the normal urinary excretions of patients in bed on average diets seldom exceed 0·1 grm., and that in *Case* 3, although the urinary output on the higher calcium diets might be regarded as within normal limits (*Table II*), the urinary output remained high when the intake was greatly reduced (*Table III*). The study of the calcium balance demonstrated the presence of abnormal calcium metabolism, therefore, in *Case* 2 by a negative calcium balance and abnormally high urinary calcium excretion, in *Case* 1 by the abnormally high urinary calcium excretion, and in *Case* 3 by the abnormally high urinary calcium excretion when on a low calcium intake.

FIG. 289.—*Case* 2. Calcium and phosphorus metabolism as represented in *Fig.* 288.

The study of the phosphorus balance (*Tables II* and *III*) showed a negative balance in *Case* 2 only, phosphorus retention occurring in *Cases* 1 and 3, even when a diet low in both calcium and phosphorus produced a negative calcium balance.

The results of the estimations after operation can be stated briefly as follows :—

The serum calcium (*Table I*) fell in all three cases following the removal of the parathyroid tumour, and the new level was considerably below the normal. In *Case* 3, where the level was not abnormally high before operation, the level after operation was about the same as in the other cases

where an abnormally high value had been found before operation. The rate of the fall varied, for in *Case 2* the level was still high on the first and third days after parathyroidectomy, and almost reached its lowest level in eight
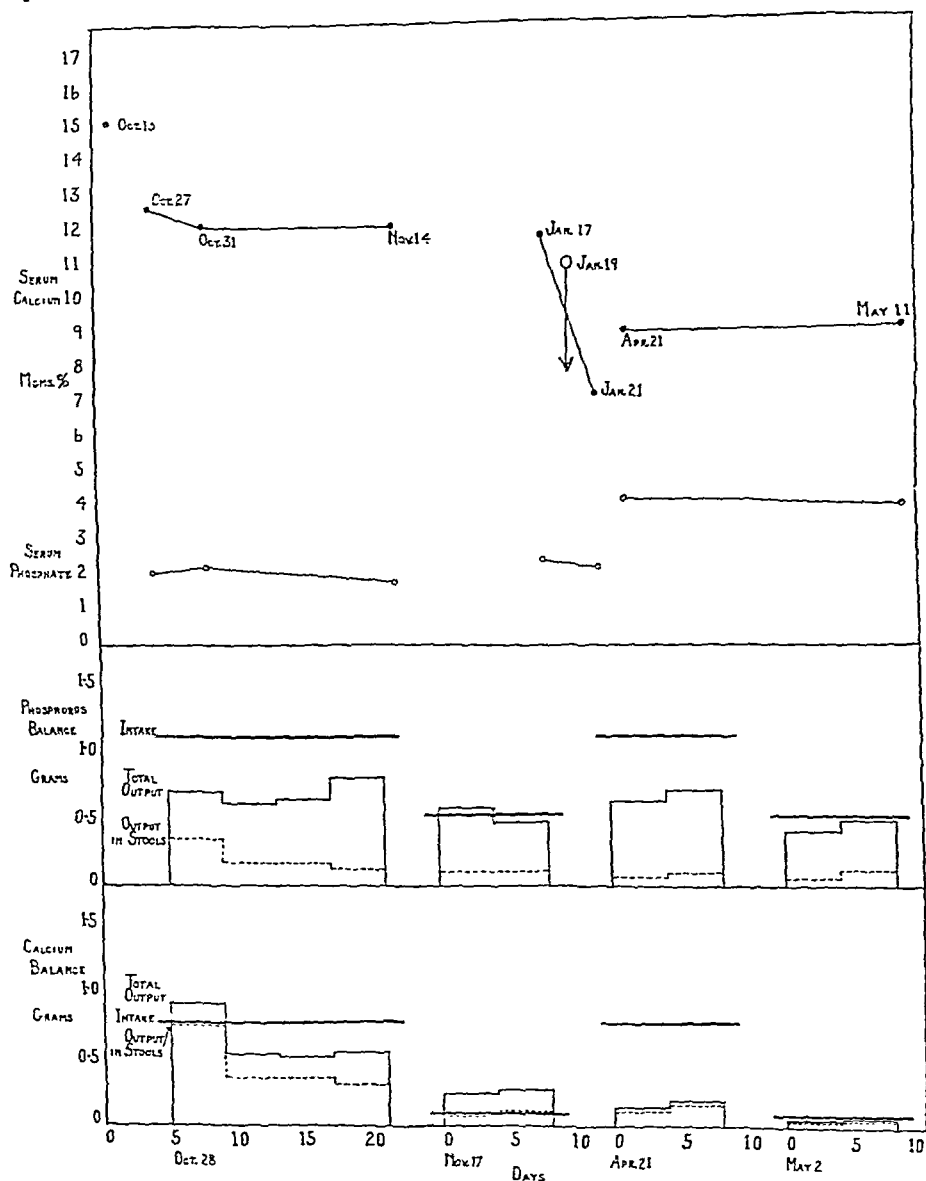
Fig. 290.—*Case 3.* Calcium and phosphorus metabolism as represented in *Fig.* 288.

days, while in *Case 3* the lowest level was reached within two days after the operation. In *Case 1* no observations were made in the days immediately following the operation.
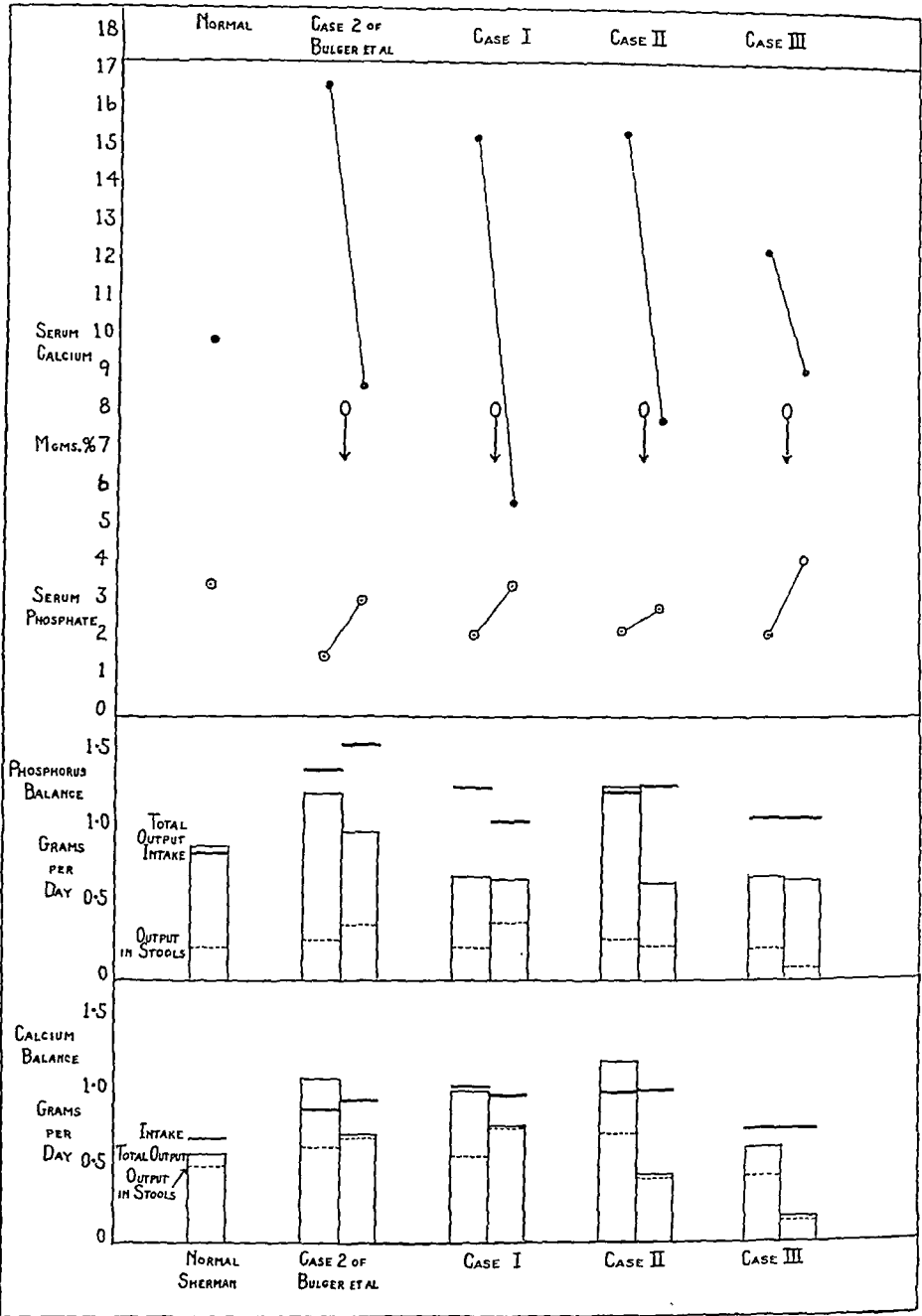
Fig. 291.—Showing the average intake and output of calcium and phosphorus before and after operation in these three cases. For comparison, a similar case by Bulger et al., and a normal calcium and phosphorus balance (Sherman), are included.

The phosphorus in the serum (*Table I*) rose after the removal of the the tumour; the rate of the increase was slow, and only in *Case* 3 had it reached a normal level within three months.

The study of the calcium metabolism (*Tables II* and *III*) showed that there was an increased retention of calcium as a result of the operation, even when there had been no demonstrable loss of calcium before the operation; the increase was greatest when a pre-operative negative balance became positive. In all three cases the excessive excretion in the urine disappeared after the operation, and the calcium excretion was mainly carried out by the intestine.

The phosphorus metabolism (*Tables II* and *III*) was little altered by the removal of the tumours in any of the cases. In *Case* 2 a slightly negative balance became positive, but the absence of any marked change in the phosphorus metabolism is noteworthy in view of the definite changes in calcium metabolism. It will be noted further that two of the patients were able to store phosphorus even while they were losing calcium. The balance data of one of the cases recorded by Bulger, Dixon, and Barr (1930), in which similar findings were made, are included in *Fig.* 291.

OPERATIONS ON
THE PARATHYROID GLANDS.

In patients with generalized fibro-cystic disease, even when the evidence is sufficiently clear to justify the assumption that a parathyroid tumour is present, this is not always easily found at operation. In none of the three cases under review did the tumour occupy a position usually occupied by a parathyroid gland (*Fig.* 292).

On examination of the region of the thyroid gland in *Case* 1, the lower pole of the left lobe was slightly more prominent than that of the right side. The operation was performed on May 8, 1930. After displaying the thyroid gland, the left lobe was dissected cleanly on its anterior and lateral surfaces. The vessels attached to its postero-lateral margin were ligated and divided to enable the lobe to be lifted and rotated towards the mid-line, and thus permit examination of the posterior surface. No tumour was found in the neighbourhood of the sites usually occupied by the superior
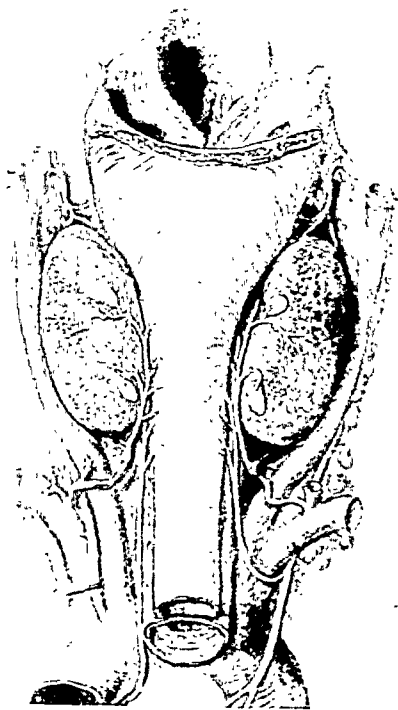
Fig. 292.—Showing the parathyroid glands on the posterior aspect of the thyroid gland (from an article by W. F. Reinhoff in Lewis's *Practice of Surgery* (1929), vol. vi).

or inferior parathyroid glands. The lower pole of the thyroid was slightly but definitely enlarged, and its consistency was such that it obviously contained a nodule. The surface in this region was smoothly continuous with that of the thyroid gland. It was considered that the enlargement might be due to an adenoma, an involution nodule, a cyst, or—conceivably—a parathyroid tumour. So little did it differ from the appearance commonly seen in a thyroid gland that it was left for the moment and the search for a parathyroid tumour continued on the opposite (right) side. This lobe was turned forward in a similar manner, and the regions of the superior and inferior parathyroid glands were examined. No tumour was found, nor was there anything abnormal in the shape or consistency of this lobe. In the absence of a parathyroid tumour elsewhere, and the almost certain knowledge that one must be present, unless the interpretation of the bone changes and the investigation of the calcium metabolism were wrong, the lower pole of the left lobe, with the nodule contained within it, was excised (*see Fig.* 293). Subsequent histological examination showed that the nodule was a parathyroid tumour (*see Figs.* 294, 295). An attack of tetany on the following day gave further confirmation of its nature.

In the second patient, *Case* 2, a slight boss was palpable before the operation on the anterior surface of the mid portion of the right lobe. This proved to be a simple cyst about 1 cm. in diameter. The sites usually occupied by the parathyroid glands were examined without revealing any tumour. A slight fullness was then seen medial to the right carotid artery. The level was about the junction of the middle and upper third of the lobe of the thyroid. On retracting the carotid sheath, and dissecting the areolar tissue over and around the prominence, a tumour gradually became defined. This was removed (*see Fig.* 296). Histological examination of this tumour showed it to be a parathyroid tumour (*see Fig.* 297). No tetany followed this operation.

The operation in *Case* 3 was performed on January 19, 1931. In this case also the parathyroid tumour might easily have been missed. The thyroid gland was of normal size and appearance. The posterior aspect of each lobe was accessible and readily examined. The carotid artery on each side lay normally and no protrusion showed such as was seen in *Case* 2. The right carotid sheath was retracted and the region behind it examined, and subsequently the left. Behind the left carotid sheath, high up, lying on the prevertebral muscles, the rounded end of a smooth, reddish-brown structure was seen. The upper end of this could not be seen even with firm retraction, and it had to be delivered by feel. The tumour was plummet-shaped and flattened (*see Fig.* 298). Microscopic examination showed this to consist of parathyroid tissue (*see Fig.* 299). An attack of tetany on the following day—and continuing for several days—confirmed the nature of the tumour.

Variability in Position. — Halsted and Evans (1907), Lorin and Cuneo (1921), and others have shown that the positions occupied by the parathyroid glands may vary within a wide range, but it was scarcely to be expected that in three successive cases the situation of a tumour arising in one of these glands should be so unusual. This may be the reason for the occasional

failure to find a parathyroid tumour when the evidence of its presence seems conclusive, as in the cases of Barrenscheen and Gold (1928), and of Richardson, Aub, and Bauer (1929).

A further point of interest in connection with the first patient is that, as the tumour was embedded in the thyroid, it must happen occasionally that a normal parathyroid will be sacrificed in the operation of partial thyroidectomy. In this patient the parathyroid, if of normal size, would not have been saved by stripping back the capsule, or shaving off the posterior border of the gland.

DESCRIPTION OF THE PARATHYROID TUMOURS.

Case 1.—

Macroscopic.—The tumour (*Fig.* 293) measures 3 × 1·5 × 1·5 cm. It is irregularly oval in shape, and its colour is yellowish red. It is encapsulated, and thyroid tissue is adherent to the capsule in several places. Fibrous trabeculæ arising from the capsule traverse the tumour, and many cystic spaces are present.

Microscopic.—The tumour (*Fig.* 294) contains obvious spaces of irregular shape, these accounting for approximately one-fourth of its total bulk. It is surrounded by a capsule which along one side and one end divides to include thyroid gland tissue. From the capsule fibrous trabeculæ enter and divide up the gland (*Fig.* 295). The gland tissue is well supplied with blood-vessels—large, small, and capillary. There are also blood-spaces
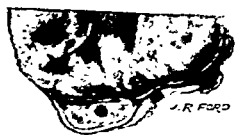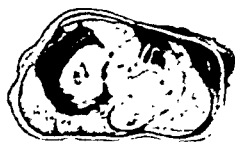
Fig. 293.—*Case* 1. External appearance and cut surface of the tumour. (*Actual size.*)
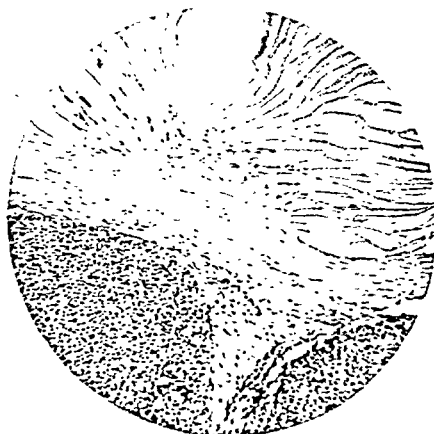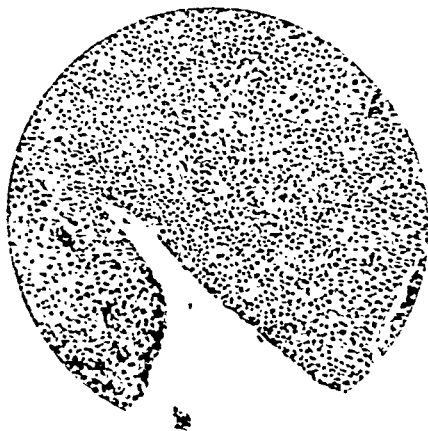
Figs. 294, 295.—*Case* 1. Microphotographs of sections of the parathyroid tumour (hæmatoxylin and eosin).

in which blood is only separated from the parenchyma of the gland by flattened cells.

The tumour cells form a fairly uniform field in that there is no suggestion of columns and no attempt at vesicle formation. The individual cells are fairly clearly defined, and vary greatly in shape and size, and many of the cells contain a double nucleus. The cytoplasm is slightly but definitely basophilic, and nowhere can the oxyphile cells of Welch—so commonly found in the normal parathyroid—be found. Many cells are vacuolated, this and the irregularity in size of the nuclei being a conspicuous feature of every field. As the vacuolation advances, the cell becomes relatively large and very clear, with the nucleus still remaining densely stained. The tumour therefore consists of the 'chief' cells of Welch in which multiplication and vacuolation are taking place throughout the tumour.

The cystic spaces appear not to have a lining membrane. Some of them contain fibrin ; towards the edges this fibrin is becoming organized, and it is continuous with the fibrous trabeculæ of the tumour. There is very little intercellular stroma, the cells being closely packed.

Case 2.—

Macroscopic.—The tumour (*Fig.* 296) measures $2\frac{1}{2} \times 2 \times 2$ cm. It also is oval in shape and encapsulated. It is yellowish in colour, the vessels showing red on the surface. On section the tumour is solid.

FIG. 296.—*Case 2.* External appearance and cut surface of the tumour. (*Actual size.*)

FIG. 297.—*Case 2.* Microphotograph of section of the parathyroid tumour (hæmatoxylin and eosin).

Microscopic.—In this tumour no cystic spaces are present. Within the capsule the character and arrangement of the cells are similar to those in the first case, and no tendency to vesicle formation is seen, but in the capsule itself some parathyroid cells are found, and some of these are arranged in single or double columns and vesicles (*Fig.* 297). No oxyphile cells are seen.

Case 3.—

Macroscopic.—The tumour (*Fig.* 298) measures 3·5 × 1·5 × 0·7 cm. Its shape is that of a flattened plummet. Its colour is dark yellowish brown. It contains much subcapsular hæmorrhage. This was probably caused by the difficulty in delivering the tumour owing to its depth combined with the high situation of its upper pole.

Microscopic.—This tumour differs from the other two in that much more stroma is present, and the cells over a wide extent tend to be grouped
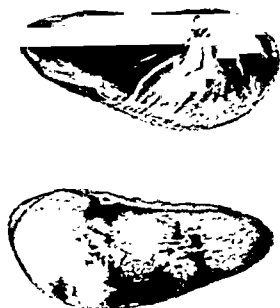
FIG. 298.—*Case* 3. External appearance and cut surface of the tumour. (*Actual size.*)

FIG. 299.—*Case* 3. Microphotograph of section of the parathyroid tumour (hæmatoxylin and eosin).

in ill-formed vesicles (*Fig.* 299). This is reminiscent of the appearance sometimes seen in fœtal adenoma of the thyroid gland. A very few oxyphile cells are present.

It will be remembered that in this patient the bone changes were more advanced and more widespread than in the other two, although the blood calcium at the time of operation was very little raised. Our knowledge is not yet sufficient to enable us to say whether the vesicular or non-vesicular appearance is associated with differences in the functional activity of the tumour. Over small areas the appearance of the parenchyma is similar to that of the other two tumours.

DIAGNOSIS.

An association of the generalized form of osteitis fibrosa with hyperparathyroidism has been shown sufficiently often to suggest that hyperparathyroidism may prove to be the cause of generalized osteitis fibrosa. If this should be so, then a diagnosis of generalized osteitis fibrosa would be sufficient to justify an operation on the parathyroid glands. In the present state of knowledge this attitude is not justifiable, and the demonstration of the presence of hyperparathyroidism should be made before operative treatment is undertaken. In each of the three cases reported here, the presence of

generalized osteitis fibrosa and also of hyperparathyroidism was demonstrated, but the grounds for the diagnosis differed in the three cases. In none of the cases was there doubt that the patient was suffering from generalized osteitis fibrosa. All had pains and aches in the limbs, spontaneous fractures, cyst-like areas of rarefaction in the bones, general rarefaction of the bones elsewhere, muscular weakness, and wasting. In one (*Case* 3), there were cellular tumours of bone, and in each of the three cases the cysts or tumours caused expansions of the bones sufficient to be recognized by clinical examination. Renal calculi were present in two of the cases.

In *Case* 2 the evidence of hyperparathyroidism was complete (Barr and Bulger (1929), Bulger, Dixon, and Barr (1930)). The serum calcium was constantly abnormally high, the serum inorganic phosphorus abnormally low, the output of calcium exceeded the intake even on a generous diet, and the urinary excretion of calcium was especially increased. In *Case* 1 the evidence was nearly as good, for the serum calcium was constantly high, and the serum phosphorus low, but calcium was being stored when the intake was adequate. The high urinary excretion of calcium was, however, so striking a feature even on a low calcium intake that the presence of hyperparathyroidism was diagnosed with confidence. In *Case* 3 the evidence was less clear. On one occasion the serum calcium was abnormally high; but during the periods of the controlled observations it was not found to be above a high normal level, although the serum phosphorus was consistently low. The balance experiments failed to show an excess of calcium output over intake except when the intake was markedly inadequate. The high urinary excretion of calcium, however, on a diet containing an inadequate amount of calcium, though not so striking as in *Case* 2, seemed, in conjunction with the other evidence, sufficient to warrant an operation on the parathyroid glands.

It is clear that difficulties may arise in the diagnosis of hyperparathyroidism unless the serum calcium is abnormally high. In *Case* 1, five years before the patient was finally treated by operation, and at a time when the generalized osteitis fibrosa was undoubtedly present, the serum calcium was normal. In *Case* 3 only one estimation out of four showed a definitely abnormally high level of the serum calcium. In such instances elaborate investigations of calcium metabolism are necessary to establish the diagnosis, and even then the faulty metabolism may not be detected unless low calcium intakes are utilized and the high urinary excretion is demonstrated. These findings suggest that even in the presence of a parathyroid tumour, the excessive activity of the parathyroid glands will vary greatly from time to time, so that investigations at any one period in the disease may fail to demonstrate the presence of hyperparathyroidism. In the three cases reported here the most constant abnormality was the increased urinary excretion of calcium.

The relation between the disease of the bones and the hyperparathyroidism is not clear, but whether the disease of the bones is secondary or primary, it would appear that the lesions in the bones will be the immediate cause of the recognition of the condition. The diagnosis of

generalized osteitis fibrosa is not difficult when the lesions are advanced, as in most of the cases hitherto reported and in our three cases, but when the lesions are less pronounced, the diagnosis must be more difficult. This difficulty is important, since we have seen that even in an advanced and typical case of generalized osteitis fibrosa with a parathyroid tumour (*Case* 3), the chemical investigations may fail to show clearly the presence of hyperparathyroidism. In the case of a fourth patient an operation was performed on the neck, but no parathyroid tumour or enlargement was found. In this case the evidence for a diagnosis of generalized osteitis fibrosa was not complete, and the chemical investigations were not indicative of hyperparathyroidism, but the possibility that it was an early case was considered sufficient to justify the exploration. The patient was a married woman, 26 years old. At the age of 9 a fracture of the left femur resulted from

Fig. 300.—Skull from case of multiple cystic disease of bones without hyperparathyroidism, showing localized thickening and irregular mottled appearance about junction of occipital and posterior parietal regions.

a trivial fall, and she had complained of pains in the left leg off and on since the accident. When admitted to hospital in December, 1930, the femora were both bowed and there was a swelling in the middle of the shaft on the left side. X-ray examination showed :—

1. *Skull* (*Fig.* 300).—Thickness of calvarium normal except for the presence of localized superficial thickening. which was most marked about the junction of the occipital and posterior parietal regions ; irregularly mottled in appearance.

2. *Left Humerus* (*Fig.* 301).—Expansion of central part of shaft with appearance as of cysts with sclerosed bone forming their walls, but cavities not so clear as in true cysts ; cortical layer on inner side of expansion thinner than in control, and bone beneath rarefied ; cortical layer on outer side thickened.

3. *Pelvis.*—Extensive rarefied areas in iliac bones, with appearance as of cysts with sclerosed margins.

4. *Left Femur.*—Expansion of neck and most of shaft; upper part of neck fractured, but arrangement of bone abnormal; lower part of neck occupied by rarefied area like a cyst and expanding bone; shaft curves outwards; cortical layer on inner and outer margins thinner than normal, and outer layer showing wavy outline towards centre of bone; large cavity, apparently cystic, occupying middle of shaft with split in external cortical layer opposite to it; similar cavity lower in shaft and several smaller ones in lower end of bone: whole medulla irregular in appearance as if occupied by less clearly defined spaces.

5. *Left Fibula.*—Expansion in middle of shaft due to fusiform expansion of medullary cavity with sharply defined upper and lower margins; cortex as thick as in control but density less.

6. Lumbar spine, bones of forearms, tibiæ, right humerus, right femur, and left fibula show no abnormalities when compared with controls.

The left femur was explored in two places. In the central part of the shaft at the site of the old fractures, large intercommunicating cavities were found surrounded by dense fibrous tissue. In the neck of the bone, at the site of what appeared to be a large cyst, there was no cavity, the area being filled with masses of firm fibrous tissue. The fibrous tissue from the two places was similar, and on microscopical examination showed no normal bone or marrow, but consisted of dense fibrous tissue well formed and very vascular. Embedded in this were many small pieces of bone, irregular in shape and consisting of woven bone without a proper lamellar structure. There was no osteoid layer and no evidence either of resorption by osteoclasts or of apposition.

Fig. 301.—Left humerus from a case of multiple cystic disease of bones without hyperparathyroidism (A)—with a control (B)—showing expansion of central part of shaft with appearance as of cysts with sclerosed bone forming their walls.

The chemical investigations showed normal values for the calcium and phosphorus in the serum, and no abnormality of calcium and phosphorus metabolism was detected except that the urinary excretion of calcium was slightly higher than that in normal controls, when on a diet providing a low calcium intake. In view of the failure to discover any abnormality in the

parathyroid glands at operation, this case may be one of focal osteitis fibrosa. but it is right to point out that the criteria, recognized at present, for a diagnosis of generalized osteitis fibrosa are based on the study of advanced cases, and that studies at different stages, and especially in the early stages of the disease, not only of the bones but also of the parathyroid glands, are lacking.*

From the observations on our three cases and the reports in the literature of other cases, generalized osteitis fibrosa can usually be distinguished from focal osteitis fibrosa on the following grounds. In either disease any bone may be affected : the general affection of the calvarium seen in the cases with hyperparathyroidism has not been recorded in focal osteitis fibrosa : the changes that have been recorded in the latter disease, as in the case without parathyroid tumour described above, differ greatly, as seen in skiagrams, from the changes seen in generalized osteitis fibrosa. Osteoporosis of the bones generally, and particularly in parts which do not show cystic changes, is characteristic of generalized osteitis fibrosa, and can usually be recognized by a diminution in the thickness of the cortical tissue or by an increase in the size of the Haversian spaces, but is best demonstrated by means of X rays by comparison with controls. The osteoporosis is not always very evident in the early stages of the disease (*Case* 2), but is at no time evident in focal osteitis fibrosa. As a rule thickening of bone is seen only in the calvarium in cases of generalized osteitis fibrosa, but was present in other bones in *Case* 3 ; in focal osteitis fibrosa the bones are usually much thickened at the site of the massive fibrosis of the marrow. Skiagrams in generalized osteitis fibrosa fail to show fibrosis of the marrow, as it is a fine fibrosis, differing from the massive fibrosis of focal osteitis fibrosa, which replaces the medullary cavity and may be recognized in skiagrams by reason of the fact that the outlines of the fibrotic mass throw visible shadows.

If the bony lesions are explored, further evidence of value in differentiation is obtained. True cysts are unusual in focal osteitis fibrosa, but have been reported ; in generalized osteitis fibrosa they are usual, and may be central, expanding the bone, or may be in the cortical layer. The cyst-like appearances seen in the focal disease are due to the replacement of the bony structures by fibrous tissue. Osteoclastomata occur as gross tumours in a comparatively small proportion of cases of generalized osteitis fibrosa. In the cases with the lesions of fibrocystic disease in a number of bones but without hyperparathyroidism, osteoclastomata have not so far been found ; in localized fibrocystic disease every stage may be found from a typical

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* Since the preparation of this article, a very early case under the care of A. F. Hurst and J. F. Venables has been operated upon and a tumour removed by one of us (T. P. D.). This case was orally reported at the meeting of the Association of Surgeons in May, 1932, and will be reported elsewhere. In this patient neither deformity nor fracture had occurred. Great muscular weakness associated with aching pains—especially in one hip region—led to an X-ray examination. Decalcification of bones appeared to be present. This was confirmed by taking controlled skiagrams of a number of long bones. Examination of the blood serum revealed a high calcium content. A parathyroid tumour was found in the middle of the right lobe of the thyroid. The pains disappeared and muscular strength returned following the operation.

osteoclastoma, through cysts with giant-cell tissue in the walls, to cysts in which only a few osteoclasts are found in the margin.

Apart from a study of the bones there are other criteria of value in the differentiation. Generalized osteitis fibrosa is twice as common in women as in men ; the onset is usually in adult life or in adolescence ; bone pain is usual apart from or previous to the occurrence of fracture ; bending of bones occurs from a fracture or collapse of a cystic area ; muscular weakness and hypotonicity are common ; renal calculi are common ; and when the onset of the condition occurs in young subjects there may be delayed general and sexual development. Focal osteitis fibrosa seems to occur equally in males and females ; the onset is usually in childhood or adolescence ; bone pain is absent or slight except when fracture occurs ; bending of bone occurs particularly in the upper ends of the femora and in the tibiæ ; muscular weakness, wasting, delayed sexual development, and renal calculi, do not occur.

SUMMARY.

1. Three cases of generalized osteitis fibrosa with hyperparathyroidism are described, with an account of the bony lesions as determined by exploratory operations and X-ray examinations, and the results of chemical investigation of the calcium and phosphorus metabolism.

2. In each of the three cases a tumour of the parathyroid glands was removed by operation, and in each case the situation of the tumour was unusual. Tetany resulted from the operation in two of the cases. A brief description is given of the naked-eye and histological characteristics of the tumours.

3. The diagnosis of generalized osteitis fibrosa depends on the demonstration of generalized rarefaction of the bones, in addition to the presence of cyst-like areas of rarefaction, cellular tumours of bone, spontaneous fractures, muscular weakness and wasting, and aches and pains in the limbs. Renal calculi were shown to be present in two of the three cases on X-ray examination.

4. An abnormally high level of serum calcium was not always found, even when a parathyroid tumour was present. In these three cases an increased excretion of calcium in the urine was constantly demonstrated.

5. In cases of generalized osteitis fibrosa the presence of hyperparathyroidism should be demonstrated, before operation on the parathyroid glands is undertaken.

6. After the removal of the parathyroid tumour the level of the serum calcium fell strikingly, and calcium was retained by the body, or, if retention was present previously, this was definitely increased.

7. An abnormal condition of the phosphorus metabolism was found in each of the three cases, but no consistent relation could be demonstrated between the changes in the calcium metabolism and in the phosphorus metabolism, either before or after the removal of the parathyroid tumours.

8. As a result of the removal of the parathyroid tumour, striking improvement in general health and in strength, and cessation of pain, resulted

in each of the three cases, and no further fractures occurred. A considerable increase in the density of the bones, especially around the cysts and in the cortical bone, was seen from ten to twenty-four months after the operation. In *Case* 2 the appearance of the skull showed a striking return towards the normal twenty months after the operation.

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CHRONIC PAROTITIS: A REPORT OF FOUR CASES WITH SIALOGRAMS.

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CHRONIC infections of the parotid gland are sufficiently uncommon to arouse special attention, and four such cases are recorded below. All four cases have been investigated by sialography, which consists of a radiographic study of the architecture of the larger and smaller ducts of the gland by the injection of radio-opaque fluids into Stenson's ducts; lipiodol is the fluid selected for injection.

The technique of sialography has recently been described by Payne,[1] who also referred to the somewhat scanty literature upon the subject, and by Pyrah and Allison[2]; the papers of these writers should be read for further details. It suffices here to say that $\frac{1}{2}$ to 1 c.c. of lipiodol is injected into Stenson's duct and a lateral radiogram is immediately taken, the head of the patient being fully extended.

FIG. 302.—Sialogram of normal parotid gland.

A normal sialogram is reproduced for purposes of comparison (*Fig.* 302).

CASE REPORTS.

Case 1.—E. V. J., male, age 36.

HISTORY.—The patient stated that in April, 1931, he suffered from an attack of influenza and immediately afterwards had a swelling of the left parotid gland. The swelling was marked during and after meals and subsided two or three hours later; when the gland was swollen he experienced some discomfort but no actual pain. During the acute stage he did not notice any discharge into the mouth. For some weeks later the swelling persisted, though it became noticeably less; but even in three or four months' time there was still a slight swelling of the gland after meals, and he then began to notice a salty taste in his mouth. Pressure over the gland caused a discharge of saliva which had a salty taste.

On Examination (Jan. 1, 1932).—There was a slight enlargement of the left parotid gland, especially of the socia parotidis. The surface of the enlarged gland was quite smooth and there was definite tenderness on pressure. The orifice of the left Stenson's duct was slightly injected, and the saliva which was squeezed out on pressure over the gland was slightly purulent. The right parotid gland was normal.

The teeth were healthy, radiological examination showing no apical disease, and the throat and nose (which were examined by Mr. E. W. Bain) were also found to be healthy. The Wassermann reaction was negative. No organisms were grown from the saliva, but pus cells were present. A sialogram of the left parotid (*Fig. 303*) showed that Stenson's duct was dilated to about four times its normal calibre but that there was no abnormality of the smaller ducts. A sialogram of the normal right parotid gland showed an exactly similar picture. Calculi in the gland were excluded by preliminary X-ray examination.

Fig. 303.—*Case* 1. Sialogram of left parotid gland. This shows an enormous dilatation of Stenson's duct and of the terminal portion of its chief contributory ducts.

Treatment.—Treatment by light massage to the gland, acid drinks to promote the flow of saliva, and mouthwashes gave no relief. Though no gross obstruction of the orifice of the gland was demonstrated, it was thought that the dilated duct resulted in some kinking at its orifice in the mouth. The terminal portion of the duct was therefore slit up for half an inch under local anæsthesia and weekly injections of lipiodol were given along the duct. The saliva is now normal and there is no salty taste in the mouth ; there is still, however, occasional fullness in the gland after meals, but the condition is much improved.

Case 2.—E. W., female, age 37.

History.—The patient stated that since July, 1931, she had had a sore mouth, particularly marked on the inside of the cheeks. A few weeks later she noticed a salty taste in the mouth and a swelling of both parotid glands. There was no appreciable increase in the size of the glands after food, but it was often considerably increased in cold weather. In December, 1931, all her teeth had been extracted without any improvement in the condition. Examination of the nose and throat revealed nothing abnormal. Since January, 1932, the patient has had a very dry mouth.

On Examination (Jan. 30, 1932).—Both parotid glands were slightly enlarged and tender. The orifices of both Stenson's ducts in the mouth were very red, œdematous, and pouting. The mucous membrane of the cheek surrounding the orifices of Stenson's ducts was the seat of an angry-looking granular stomatitis, associated in places with deep furrows evidently due to submucous fibrosis. The mucosa also showed yellowish-white areas of fibrosis (*Fig. 304*). The tongue was normal. There was a discharge of muco-pus from both parotid glands upon pressure over them. The Wassermann reaction was negative. Sialograms of both parotid glands showed very slight but definite dilatation of the smallest ducts (*Fig. 305*).

Treatment.—The chronic stomatitis improved very considerably by treatment with glycerinum boracis and hydrogen peroxide mouth-washes. The flow of saliva was promoted by regular massage to the gland and by acid drinks, and the general

Fig. 304.—*Case* 2. Appearance of mouth. There is an area of chronic granular stomatitis around the orifice of the right Stenson's duct, with much submucous fibrosis.

Fig. 305.—*Case* 2. Sialogram of right parotid gland. There is slight dilatation of the smallest ductules.

condition of the patient was improved by tonics and cod-liver oil. Weekly injections of lipiodol along Stenson's duct were given. The stomatitis almost disappeared, leaving only the furrows and fibrosis mentioned above, and the discharge of purulent saliva has now completely stopped.

Case 3.—O. B. S., female, age 47.

HISTORY.—The patient had an attack of acute right parotitis in April, 1918. The swelling, which was considerable, subsided in about a fortnight, but a few weeks later she noticed a discharge into the mouth of thick purulent mucus, coming, apparently, from the right parotid gland; this discharge ceased in a few weeks' time. In 1927, following an attack of influenza in which the tonsils were somewhat swollen, the right parotid gland again became slightly swollen and painful. In 1930 the patient had a third attack of acute parotitis of the right gland, followed a fortnight later by a similar change in the left parotid gland; these attacks were associated with swelling, considerable pain, trismus, and with slight elevation of temperature. After this third attack the patient complained of very marked dryness of the mouth and a mucopurulent discharge from both parotid glands; these symptoms have persisted ever since. The patient has had her tonsils enucleated; they were heavily infected with a hæmolytic streptococcus. She has had prolonged treatment with vaccines prepared from cultures from the tonsils and from the parotid (which also yielded a hæmolytic streptococcus). She also had the right antrum washed out. Stenson's duct has been repeatedly irrigated with a weak solution of iodine.

FIG. 306.—*Case 3.* Sialogram of right parotid gland. Shows an incomplete filling of the ducts due to fibrosis-atrophy of the gland.

ON EXAMINATION (February, 1932). — The parotid glands were not enlarged. On pressure over the right gland and to a less extent over the left a small amount of bubbly muco-pus could be expressed; microscopic examination of the discharge shows the presence of pus cells. The orifices of Stenson's ducts in the mouth are prominent and slightly red. There are no foci of infection in the teeth or in the mouth. A sialogram shows incomplete filling of the smaller ducts of the parotid evidently due to advanced fibrosis (*Fig.* 306).

TREATMENT.—The patient could not tolerate acid drinks owing to the extremely dry mouth, but evacuation of the ducts has been promoted by massage to the glands. A prolonged course of lipiodol injections, at first at weekly intervals and later every two or three days, has been given, but no improvement has taken place.

Case 4.—M. H., female, age 16. This case has been reported elsewhere, but is recorded here again to complete this series.

HISTORY.—The patient first came to the Leeds General Infirmary early in 1929 with a discharge of pus from the left ear, and returned a few months later with a swelling of the left parotid gland, which was followed a few weeks afterwards by a swelling of the right gland. At first the glands were but slightly swollen, but in July the right parotid became acutely painful, hot, and swollen; similar acute changes occurred in the left gland a few days later. There was no elevation of temperature. The inflammation in both glands subsided in a few days after

treatment by fomentations and hot mouth-washes. During the following five years both glands have been persistently larger than normal and there have been acute exacerbations every few months. The patient can at any time squeeze from the glands a considerable amount of ropy muco-pus, which, especially during the acute crises, has a very offensive taste.

ON EXAMINATION (January, 1930).—Both parotid glands were enlarged and their surfaces were lobulated throughout ; there were no signs of inflammation, but the glands had a fibrous consistency. On pressure over a gland a mixture of clear saliva and ropy mucus could be squeezed from Stenson's duct, the orifice of which was very prominent and slightly injected. On examination during an acute attack some weeks later the glands were considerably swollen and their surfaces were smooth. By gentle massage of the gland saliva containing plugs of pus mixed with epithelial débris could be squeezed out of Stenson's ducts in considerable quantity.

FIG. 307.—*Case* 4. Sialogram of right parotid gland. Shows enormous dilatation of the smallest ductules.

The acute attacks lasted from one to three weeks. The teeth were very unhealthy and showed a deficiency of enamel, many having broken off near the alveolar margins while all the remainder were carious. The throat and tonsils were healthy ; the discharge from the ear subsided three years ago. The blood-count was normal and the Wassermann reaction was negative. X rays revealed no parotid calculus. A culture of the débris discharged from the duct gave a growth of a green - forming streptococcus; the débris itself consisted of pus cells in large quantity, mucus, and desquamated epithelial cells. Sialograms of both glands showed a very marked dilatation of the terminal alveoli; the extent of this dilatation was uniform throughout the gland, and the picture resembles that of bronchiectasis of the lungs (*Fig.* 307).

TREATMENT.—All the teeth have been removed. The condition is showing gradual and marked improvement after prolonged massage to the glands and the use of acid drinks and mouth-washes and occasional injections of lipiodol into Stenson's duct. The girl's general condition has been considerably improved by artificial sunlight, tonics, malt extracts, and by short periods of convalescence in the country ; she now appears to be progressing towards a complete recovery.

COMMENTARY.

Pathology.—If the specific chronic infective diseases of the parotid gland are excluded—namely, syphilis, tuberculosis (including uveo-parotitic fever), actinomycosis, and the condition known as Mikulicz's disease—there remain a number of non-specific infections of the gland which have probably a varied etiology.

Cases 2, 3, and 4 above are examples of chronic parotitis arising from infective foci in the mouth. In *Case* 2 a chronic stomatitis had preceded the

parotitis and the two conditions subsided synchronously after treatment. *Case* 3 had had an infection of the tonsils (by a hæmolytic streptococcus) immediately preceding the initial acute attack of parotitis, while in *Case* 4 the parotid infection probably arose from carious teeth.

In *Case* 1 the parotitis possibly resulted from a blood-borne infection, but it is equally possible that the infection ascended Stenson's duct from the mouth, which, during the febrile stages of the influenzal attack, is dry and likely to be harbouring pathogenic organisms. The great dilatation of Stenson's duct is of interest in this case ; as it is bilateral it is probably congenital. The case report indicates that there seemed to be a definite obstruction near the orifice of the duct, the lumen of which is quite four times that of a normal Stenson's duct ; once free drainage of the duct had been provided, the infection rapidly disappeared. A similar case of great dilatation of one Stenson's duct was reported by Barsony.[3] Such a condition must be very rare.

There is no doubt that in some cases of chronic swelling of the parotid gland the infective lesion lies in the ducts. Raymond Johnson[4] reported such a series. The patients had a unilateral painful enlargement of the parotid gland which became larger after food. Pressure over the gland caused the extrusion of a plug of mucus from the duct followed by a flow of watery saliva ; this gave a temporary relief of symptoms. The orifice of Stenson's duct in the mouth was red and inflamed. The swellings usually disappeared after some months of treatment, but occasionally they reappeared at intervals.

Kussmaul[5] described some cases of chronic parotitis in adults which he called 'sialodochitis fibrinosa'. In half of his cases the condition was bilateral. The parotid gland became suddenly swollen and the patient had considerable pain, trismus, and in some cases fever. The condition was relieved after some hours, or days, by the extrusion from Stenson's duct of a long plug of fibrin ; following the fibrin, a stream of saliva escaped and the swelling of the gland subsided. Kussmaul compared these cases with bronchitis fibrinosa.

No biopsies have been possible in any of the four cases reported, but the sialograms throw some light upon the pathology. The normal architecture of the ducts of the parotid gland is shown by the normal sialogram, which is a typical one taken from a number of normal ones. The sialogram of *Case* 2 shows the earliest deviation from the normal picture ; here Stenson's duct is of normal size, but many of the terminal ductules show small dilatations while others are perfectly normal ; the disease in this case subsided in about nine months.

The sialogram from *Case* 4 shows a much later stage in the disease ; here, after four and a half years of chronic infection, the terminal ductules are so dilated that they appear on the sialogram as spheroidal spaces and they resemble perfectly the dilatation of the terminal bronchioles in bronchiectasis of the lungs. The large dilatations of the terminal ducts allow of the accumulation of much mucopurulent débris. and, while they are probably the result of infection. they also serve to perpetuate it indefinitely. Both Keith[6] and Payne[1] have recorded sialograms of similar cases.

The sialogram of *Case* 3 shows quite a different result of chronic long-standing infection. Although exactly the same syringe and technique were

employed as for the other sialograms, the filling of the terminal ductules was for the most part incomplete, though here and there very small dilatations can be seen; the gland in this case has evidently undergone very advanced fibrosis as a result of the long-standing infection, and this fibrosis is reflected clinically in the extreme dryness of the mouth from which the patient suffers.

Calculi were present in the parotid glands of none of the four cases. Though uncommon, calculi do accompany a number of cases of chronic parotitis; a recent series has been recorded by New and Harper.[7]

Symptomatology.—All four cases have had an acute or subacute onset, but none has proceeded to the stage of suppuration. During the acute stage the gland has been hot, tender, and painful. One case (Case 4) has had repeated subacute exacerbations which mimicked the first attack. Similar cases of recurrent parotitis have been described by Walker,[8] Payne,[1] and by Hobbs, Sneierson, and Faust.[9] During the chronic phase of the disease there was either no discomfort at all or a little fullness of the gland after meals. Throughout the disease pus in smaller or larger amounts is discharged from Stenson's duct.

It is of interest here to note the occasional occurrence of aerocele of the parotid gland, and a case has very recently come to my notice; such a condition may mimic subacute infections. The patient, a girl of 10, habitually had a glass of milk in the evenings, and sometimes she took the milk from the glass with an exaggerated sucking action of the mouth. On several such occasions the parents noticed an acute enlargement of both parotids which rapidly diminished upon cessation of drinking and upon gentle massage of the gland. On one occasion the father, who is a medical man, noticed that synchronously with the swelling of both parotids an extensive surgical emphysema of the cheek and side of the head had developed. The provisional diagnosis of aerocele of the parotid gland was thereby confirmed; the emphysema must have developed from a tiny tear in Stenson's duct or in a smaller duct resulting from the pressure of air aspirated into the gland. A similar case of acutely developing parotid swelling in a baby was described by Wolff (cited by Steinitz[10]), and he suggested that the cause of the swelling was a spasm of the orifice of Stenson's duct; the case, however, was probably one of aerocele of the parotid gland.

Treatment.—Rational treatment consists in the treatment or the removal of foci of infection in the mouth, in the promotion of secretion from the gland in order to ensure evacuation of the purulent débris which accumulates in the ducts, and in irrigation of the ducts with some mild non-irritating antiseptic.

The treatment of infective foci in the mouth may be assisted by the administration of vaccines prepared from cultures of the organisms in such foci; in the solitary case in which this method has been tried the patient derived no benefit (Case 3). In Case 2 the improvement of the parotitis ran parallel to the improvement of the stomatitis.

Promotion of drainage of infective material from the ducts is best obtained by gentle massage to the gland; this can be done, after instruction, by the patient himself, and consists in slow oft-repeated pressure with the

fingers moderately firmly over the gland from behind forwards. The patient can easily perform the treatment several times daily. The flow of saliva can be increased by acid drinks (lemonade, etc.) throughout the day and by the frequent use of chewing gum. Calculi in the gland or in the duct should be removed and cicatricial narrowings in the duct should be dilated or divided. Hobbs, Sneierson, and Faust[9] have carried out dilatation of the orifices of Stenson's duct for a series of cases of acute and chronic parotitis, using filiform bougies. Most cases of acute parotitis subside without such treatment, and it is difficult to see what it can offer in the absence of a stricture of the duct.

The instillation of $\frac{1}{2}$ to 1 c.c. of lipiodol into Stenson's duct has proved successful in two of the cases of parotitis reported above. Lipiodol is non-irritating, contains about 50 per cent of iodine, and, being a highly viscid oil, remains in the ducts for several hours before it is discharged. Payne also has found this method of treatment very useful. The injections are made at weekly intervals, and for short periods injections may be given on alternate days. Hobbs, Sneierson, and Faust[9] used injections of saline and of a solution of 1 per cent mercurochrome.

SUMMARY.

1. Four cases of chronic parotitis are reported, and the sialograms of these cases are shown.

2. The pathology of the cases is discussed; and the uses of sialograms in elucidating the morbid anatomy of the infected glands and of their ducts are demonstrated.

3. The treatment of chronic parotitis is outlined.

I desire to thank the members of the Honorary Surgical Staff of the Leeds General Infirmary for permission to record these cases which have been under their care.

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THE TECHNIQUE OF RESECTION OF THE PRESACRAL NERVE (COTTE'S OPERATION).

By A. A. DAVIS, MANCHESTER,

DICKENSON RESEARCH SCHOLAR IN SURGERY.

SINCE Cotte, of Lyons, first described his operation in 1925, in his now classical article in the *Journal de Chirurgie*,[1] the procedure has been widely adopted, more especially in the Latin countries, for the relief of a variety of painful pelvic conditions, particularly gynæcological. Aievoli,[2] Aubert.[3] Bernard and Theodoresco,[4] Chianello,[5] Cotte,[6, 7, 8] Derom,[9] Ferey,[10, 11] Georgesco,[12] Gomoiu,[13] de Grisogono,[14] Hallopeau.[15] Heitz,[16] Learmonth and Braasch,[17] Mornard,[18] Michon,[19] Paolucci.[20] Petersen-Ekkert,[21] Spirito,[22, 23] Tirelli,[24] Viannay,[25] and Walther[26] have all reported good results, and although a certain amount of exaggerated enthusiasm has undoubtedly been exhibited by its more ardent advocates, there is no doubt that the operation is definitely establishing itself as one of the most successful in sympathetic surgery. It is as yet too early adequately to assess the results in the twenty cases operated upon, with Dr. W. R. Addis, during the past year at the St. Mary's Hospital, Manchester, but in view of the rapidly increasing popularity of the operation among English surgeons, it is suggested that a description of certain technical details and difficulties learned in its performance might be of service. (It is of course desirable, here as elsewhere, that cadaveric experiment should precede any actual operation.)

TECHNIQUE OF THE OPERATION.

Continental opinion is divided between Pfannensteil's transverse suprapubic incision and a median subumbilical one, with a bias in favour of the latter. We ourselves employ a left paramedian incision, sliding the rectus outwards. A length of from 3 to 4 in. from the umbilicus downwards is usually adequate, though in fat patients this may have to be prolonged upwards. Here, as elsewhere, æsthetically designed incisions occasionally contrive to produce a certain amount of internal difficulty ; and as in these cases one is working at considerable depth, intestinal trauma can be avoided only by a reasonable exposure.

The peritoneum opened, the abdominal and pelvic organs are rapidly examined, and any abnormality is dealt with. (We have never considered it justifiable to omit the surgical treatment of a pelvic organ found requiring it, as advocated by certain French writers, in order to assess the value of presacral resection alone.) This done, the Trendelenburg position is adopted, and the site of operation exposed by careful packing off of the bowel on all sides. This is best accomplished with a single roll of 8-in. gauze, relaxation being much facilitated by the employment of spinal anæsthesia.

The 'three points of Cotte' should next be definitely identified by palpation. They are : (1) The promontory of the sacrum ; (2) The bifurcation of the aorta ; (3) The inferior mesenteric artery. In this connection it should be remembered that the 'presacral nerve' of Latarjet is neither presacral, nor is it usually a single nerve. In the majority of cases it takes the form of several communicating filaments lying on the anterior surface
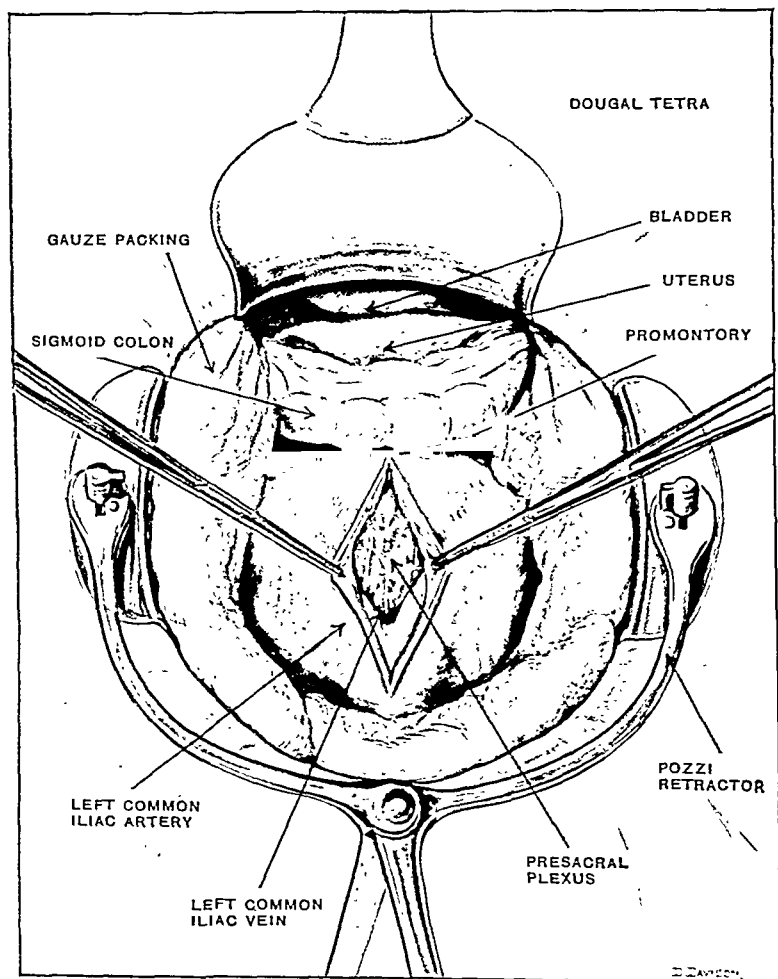
FIG. 308.—Resection of the presacral nerve : the operative field.

of the 5th lumbar vertebra, and must be sought for in this position. The nerves can occasionally be seen in thin subjects, shining through the peritoneum, and can usually be palpated through it.

The posterior parietal peritoneum over the nerves is lifted gently and snipped with scissors, which continue the incision upwards and downwards for an inch or more, exposing the operative field as depicted in *Fig.* 308.

No attempt should be made to dissect out and excise the individual nerves, as this is usually impossible, and is in any case anatomically inadequate. Instead, the layer of fibrocellular tissue in which the nerves run, and which lies between the posterior parietal peritoneum anteriorly, and the left common iliac vein and 5th lumbar vertebra posteriorly, should be systematically removed in one sheet, from the left common iliac vein to the right common iliac artery laterally, and from the aortic bifurcation above to the sacral promontory below. A little over one inch of the nerve or nerve plexus is thus exsected. It is unnecessary to transgress these limits, particularly below the promontory, where the 'carrying tissue' blends with the sacral periosteum, allowing of easy rupture of the middle sacral artery, which in this situation is nearly inaccessible to ligature.

The fascia containing the presacral nerves is of a peculiar density and toughness, and any doubt of its validity is easily dispelled by firm traction exerted upon its proximal cut end.

Cotte has suggested, on the basis of his findings in cases of recurrence operated upon a second time, that an occasional cause of failure to relieve or prevent reappearance of the pain is probably the presence of a neuroma at the cut end of the nerve, analogous with the traumatic neuroma of the peripheral spinal nerves, and secondary to ligature irritation. Leriche and Fontaine[27] have demonstrated their occurrence experimentally, and Bewerschenko[28] has suggested a variety of heroic treatments for its prevention, but probably the most reasonable way of accomplishing this is the *entire omission of ligature* in separating the resected nerve. Hæmorrhage is usually negligible, coming solely from the tiny vasa nervorum, and is controlled by tampon pressure. The occasionally brisk arterial bleeding from an abnormal vessel is easily seen and clipped.

A continuous peritoneal suture of No. 90 linen thread completes the intra-abdominal portion of the operation, and the wound is closed in layers.

DIFFICULTIES AND DANGERS.

Perusal of the literature reveals an almost monotonous repetition of statements declaring the ease and safety of the operation, but even our limited experience has convinced us that these must be somewhat optimistic. It is true that, given a thin subject exhibiting no anatomical abnormality, resection of the presacral nerves may be easily and rapidly performed (although even in the simplest cases the proximity of the great vessels calls for the exercise of considerable caution). There remains, however, a large minority of cases in which some difficulty must be experienced, and it is proposed to enumerate these as we have encountered them.

1. The presence of much fat in the abdominal wall may so increase the depth of the operative field as to make manipulation very difficult. An adequate incision, combined with the highest Trendelenburg position, facilitates matters.

2. The presence of a pelvic tumour may entirely negative the procedure. If simple, it should be removed as a preliminary. If inoperable, resection of the *intermesenteric nerves*, as suggested by Bernard and Theodoresco[4], may be performed.

3. The pelvic mesocolon, normally confined to the left of the mid-line, may transgress it, thus lying across the field of operation. Roussel[29] found this abnormality in 15 per cent of his anatomical subjects, but this incidence is probably much too high, and in either case its presence is not, as Roussel suggests, a complete bar to presacral resection. Where this difficulty is encountered, it is necessary to incise both leaves of the mesosigmoid in order to expose the peritoneum covering the nerves. The mesosigmoid should be traversed parallel with its root, and half an inch from it, to avoid the superior hæmorrhoidal artery. Dissection between the layers should be carefully avoided, as it confuses localization.

4. We have seen an accessory ureter running in the mid-line on three occasions, closely simulating a single large presacral nerve. It may be identified by palpation and the effect of stimulation.

5. In a case of abnormally low bifurcation of the aorta the inferior mesenteric artery was found running vertically downwards in the mid-line. The nerves in this case were resected piecemeal on either side of the artery.

6. Hæmorrhage from the middle sacral artery is the result of a too zealous denudation of the fibrous tissue covering it as it lies on the 5th lumbar vertebra. It is unnecessary to remove this fascia, from which the superjacent nerve-bearing tissue is easily and definitely separable by blunt dissection. Under no circumstances should the resection transgress the promontory, as rupture of the vessels is then almost inevitable, and subsequent hæmostasis extremely difficult ; it is best accomplished by under-running.

7. In a case of pelvic neuralgia secondary to inoperable carcinoma of the cervix uteri (operated upon by Professor Dougal) a single nerve, much enlarged by malignant infiltration, was found only after a long search, adherent to the elevated parietal peritoneum. This occasional cause of difficulty is best surmounted by the routine preliminary stripping of the under surface of this peritoneum, as suggested and employed by Addis, of Manchester.

8. Lastly, extreme gentleness is necessary in stripping the left common iliac vein if damage to its very friable walls is to be avoided. The hæmorrhage caused in this way in one of our cases was fairly easily controlled by lateral ligature.

SUMMARY.

The technique of the operation of resection of the 'presacral nerve' is described. It is usually easy and safe in execution, but the presence of occasional abnormalities calls for the exercise of considerable caution in its performance. A description of these difficulties, as they have been observed in twenty consecutive cases, is given, together with suggestions concerning their treatment.

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SHORT NOTES OF
RARE OR OBSCURE CASES

DIVERTICULUM OF CÆCUM.

By R. KENNON, Liverpool.

Mr. E., aged 26, was seen on the third day of an attack of pain, tenderness, and rigidity in the right iliac fossa, with slight temperature. A retrocæcal appendicitis was diagnosed.

At operation an inflammatory mass was discovered behind the cæcum 3 in. by 2 in. in extent. On turning the cæcum inward a pale blue flaccid

Fig. 309.—Diverticulum of cæcum.

appendix presented itself, obviously normal. This was removed. On further palpation of the retrocæcal mass, the finger entered what was considered to be an ulcer crater $\frac{1}{2}$ in. in diameter with edges heaped up and $\frac{3}{4}$ in. broad. Under the mistaken diagnosis of tuberculosis or malignant disease the ileo-cæcal angle was resected.

Professor Dible, who examined the specimen (*Fig.* 309), reported " A very beautiful diverticulum. The muscle is defective and the floor consists

of fibrous tissue. There is a marked inflammatory change involving more especially the narrowed ostium."

The lumen admitted the little finger for 1½ in. The patient was alive and well a year later.

A DOUBLE GALL-BLADDER OPENING BY TWO CYSTIC DUCTS INTO THE COMMON BILE-DUCT.

BY R. KENNON, LIVERPOOL.

MR. R., aged 69, was first operated upon by me in 1930 for acute suppurative cholecystitis. Several large stones were removed and drainage was established. Delirium prior to operation and a known cardiac arrhythmia reduced operative procedure to the minimum. The anomaly was not recognized.

After a year of good health, painless febrile attacks associated with mild icterus supervened. With some hesitation, owing to the condition of the heart, it was decided to explore the common bile-duct in May, 1932. The
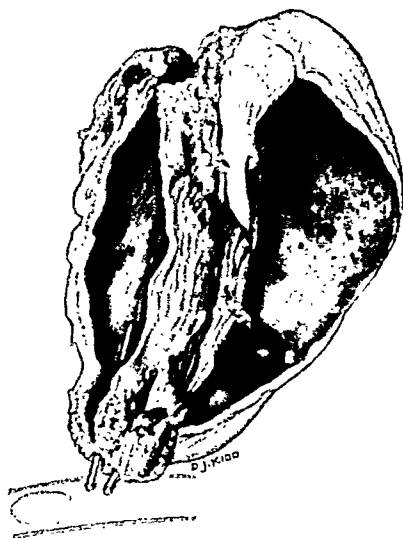
FIG. 310.—A double gall-bladder opening by two cystic ducts into the common bile-duct.

wider exposure revealed the presence of two gall-bladders—the upper one, 5 in. long and 1½ in. in diameter, containing a faceted stone : and parallel to it on its lower side, a small gall-bladder with thicker walls, 3 in. long and ¾ in. in diameter, free from stones, but with a scarred fundus, the result of previous operation (*Fig.* 310). Each gall-bladder opened separately into the common bile-duct, which contained several stones and débris.

Cholecystectomy with drainage of the common bile-duct was performed. Auricular fibrillation and a pulmonary embolus complicated a rapid local convalescence. Recovery eventually took place.

A CASE OF FACIAL PARALYSIS TREATED BY FASCIAL GRAFTS.

By R. BROOKE,

ORTHOPÆDIC SURGEON, ROYAL WEST SUSSEX HOSPITAL.

THE treatment of permanent facial paralysis due either to acute mastoid disease or to post-operative trauma is a problem which has exercised the ingenuity of the surgeon to the utmost; and it must be admitted that so far no method has been devised which deals with this complication in a satisfactory manner.

Nerve anastomoses are beset with technical difficulties, and even in the hands of the expert these operations on occasions fail. When they are successful the combination of voluntary movements with the movements of the face is extremely irksome to the patient and tends to mitigate and spoil an otherwise perfect functional result.

Section of the cervical sympathetic trunk is useful in dealing with the inflammation of the eye from which these patients so often suffer, by allowing the eyelids to come together when the levator palpebræ superioris is relaxed. It has the disadvantage that it does not overcome the facial asymmetry or improve the appearance of the mouth. In young people, especially young women, this is important, although to the patients themselves the greatest disability, and the thing about which they complain, is not the deformity, but the pain and interference with vision owing to the exposure of the eyeball to the air.

The following case illustrates a method of treating this disability by means of fascial strips which are obtained from the thigh, and are used to draw together the eyelids and elevate the angle of the mouth.

The patient, Mrs. C., aged 48 years, was first seen on Feb. 8, 1932, when she gave a history of complete right-sided facial paralysis following acute mastoid disease three years previously. She complained of pain in the right eye and interference with vision.

OPERATIVE DETAILS.—Using a modification of the method described by Passot, strips of fascia lata, approximately ¼ in. wide and 12 to 14 in. in length, are obtained from the thigh. A small vertical incision ¼ in. in length is then made slightly above and in front of the ear at the level of the commencement of the helix. This incision is situated in the hair-bearing area of the scalp, which is prepared for operation by cutting short the hair in this region in preference to shaving, as the latter may give rise to a local furunculosis.

In the depths of the wound the superficial temporal aponeurosis and the upper margin of the atrophied auricularis anterior muscle are identified. An ordinary malleable metal probe is then taken, and through the flattened eyelet end a fascial strip is threaded. The probe, eyelet end forward, is then introduced into the wound and pushed towards the inner canthus of the eye. No force is required for this, and the probe can be guided quite

readily beneath the skin along the upper eyelid. As it is malleable, it adapts itself in its passage to the conformation of the face.

A small curved incision ¼ in. in length is next made on the side of the nose half way between the inner canthus of the eye and the dorsum nasi, at the level of the inner canthus; and the lateral border of the atrophic and fibrotic procerus muscle identified. It is better to make this incision at the side of the nose rather than near the inner canthus. A wound at the inner canthus is mechanically more difficult to close and more liable to become infected by tears from the eye.
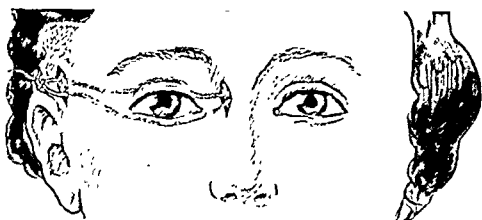
FIG. 311.—Fascial strips passed round eye to approximate eyelids.

The eyelet end of the probe is then delivered through the inner wound, unthreaded, and the end of the fascial strip drawn out. The probe is then withdrawn through the original incision and reintroduced, this time passing to the inner canthus beneath the skin of the lower eyelid near its margin until the eyelet end once more projects through the incision at the inner canthus. Part of the procerus muscle and aponeurosis are then freed from their attachment to the nasal bone, and round this the fascial strip is passed. Passot advises isolating the internal palpebral ligament and passing the fascial strip round this. If this is done, there is a risk of interfering with the tear ducts, and the resulting epiphora greatly increases the risk of infecting the wound.

The strip is then rethreaded on to the probe, and drawn back beneath the skin of the lower eyelid to the original incision. The two ends of the fascia which project from the wound are drawn tight enough to close the eyelids. Part of the fibrotic auricularis anterior and aponeurosis are isolated, and round this strips are passed and tied. (*Fig.* 311.) The incisions are closed with fine interrupted horse-hair stitches.
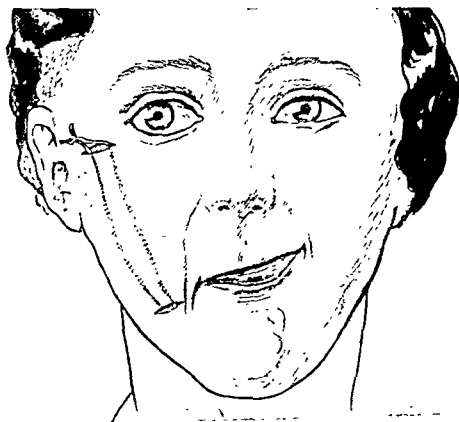
FIG. 312.—Fascial strips passed to angle of mouth to draw up the depressed corner.

The mouth on the affected side next receives attention. The fascial strip used to elevate the corner of the mouth can be introduced through the original incision made near the front of the ear, but in practice better results are obtained if a separate incision ¼ in. in length is made rather farther forward over the zygomatic process, midway between the outer canthus of the eye and the external auditory meatus. Through this incision the remains

of the zygomaticus are identified. The second incision is horizontal at the level of the angle of the mouth and should be at least ¼ in. away from the angle of the mouth, as infection of this incision will readily occur if it is placed too close to the lips. In the depths of the incision the atrophied

FIG. 313.—The patient before operation.

FIGS. 314, 315.—The patient after operation.

risorius muscle will be seen. The fascial strip is passed in this case from the upper incision down to the lower one, round part of the risorius muscle and fascia, and finally back again to the original incision, where the ends are drawn tight and tied round the remains of the zygomaticus muscle, which serves as an anchor. (*Fig. 312.*)

The degree of tension in elevating the mouth should be such that the drooping of the angle is over-corrected, especially if the operation is performed under general anæsthesia.   In the case illustrated (*Figs.* 313–315), although at the time of the operation the angle of the mouth appeared to have been raised sufficiently, when the patient had recovered from her anæsthetic it was seen that the elevation was not quite enough.   It may be impossible to identify the atrophied facial muscles in the depths of the wound, in which case the fascial strips can be anchored to parts of the aponeurosis.

The advantage of this operation over those previously mentioned for the treatment of facial paralysis lies in its simplicity.   It may be performed quite easily with local anæsthesia, and needs no special technique or detailed anatomical knowledge.   It is much less dangerous than either of the other operations.   Especially does this apply to the operation on the cervical sympathetic trunk, where the proximity of the carotid sheath and the vagus nerve is a potential danger even in the most skilled hands.

The method is open to the criticism levelled at all fascial operations, that in time the fascia will stretch.   It can only be said that these fascial strips live, and it would not seem unreasonable to suppose that they retain the properties of the fascial band from which they were obtained.   In this instance the operation is of too recent a date for any useful observations, but so far, after three months, there are no signs of any relapse, the patient has lost the pain in the eye, and has noticed a distinct improvement in her vision.

SPONTANEOUS RUPTURE OF THE SPLEEN.

By. J. M. BLACK,

SURGEON, DUNFERMLINE AND WEST FIFE HOSPITAL, DUNFERMLINE.

ACCORDING to Hamilton Bailey[1] there are only eleven cases of spontaneous rupture of the normal spleen on record, and the following case therefore seems to be worth reporting, if only on account of the rarity of the condition.

The patient, a married woman of 52, was admitted to the Dunfermline and West Fife Hospital on Nov. 18, 1931, with a diagnosis of strangulated left femoral hernia, for immediate operation.

HISTORY.—She was too ill on admission to give any history, but had had no vomiting.   After recovery she was able to give the following history. For about three years she has had a small swelling in the left groin, and on the night prior to admission she felt pain in the left groin and went to consult her doctor, who told her to rest.   For several weeks prior to this she had had an uneasy feeling in the left side just below the heart, and she thought she was suffering from heart disease.   She is quite definite that she never had a knock or any other injury to her left side.   She was up and doing her housework as usual on Nov. 18, when at mid-day she turned very sick and faint with pain in the left hypochondrium, and collapsed.   She had suffered from

no previous illness of note. She has had a family of seven, the eldest being 31 years old. There have been three miscarriages. Menstruation ceased in 1929. The Wassermann reaction was negative.

ON EXAMINATION.—The patient was a well-nourished woman of the maternal visceroptotic type. She was in an extreme state of collapse, with a subnormal temperature and a pulse of 140 per minute, and was very restless. The abdomen was slightly distended and moved well on respiration. There was tenderness all over the left side of the abdomen, maximum in the left iliac region. There was a small hard nodule in the region of the left femoral canal, not tender, with no impulse on coughing, and dull on percussion. There was dullness in the left flank on percussion.

DIAGNOSIS.—Obviously the patient was suffering from an internal hæmorrhage, and in spite of her age the most likely diagnosis appeared to be ruptured extra-uterine pregnancy.

OPERATION.—Operation was performed immediately under local infiltration with novocain and adrenalin. A left gridiron incision was made. On

FIG. 316.—Spleen showing ruptured capsule and subcapsular hæmatoma.

opening the peritoneum dark fluid blood exuded in large quantity. The uterus was rapidly palpated and was small and involuted ; the ovaries and tubes were normal. The great omentum was normal, no evidence of torsion being found.

Light ether anæsthesia was then begun and the splenic area palpated. Immediately there was a copious flow of blood. The incision was extended upwards paramedially, to the left costal margin, and the splenic pedicle was clamped and the spleen removed (*Fig.* 316). It was lightly adherent to the diaphragm by blood-clot, and the left subphrenic space was full of clot and fluid blood. The splenic pedicle was tied with catgut, and the abdominal incision was closed with through-and-through silkworm gut stitches. Pressoplast lace dressing was applied.

One pint of gum-saline and glucose was given intravenously before the

patient left the table, and two hours after operation 250 c.c. of citrated blood was administered, her husband being found a suitable donor.

She recovered from the operation, and, apart from a slight left-sided pleurisy and an attack of epistaxis, her convalescence was uneventful.

SUBSEQUENT PROGRESS.—A blood-count done on Dec. 9, 1931, showed :—

Red blood-corpuscles	.. ..	3,600,000
White blood-corpuscles	.. ..	6000
Differential Count.—		
471 cells		
		Per cent
Neutrophils	.. ..	51·9
Lymphocytes	.. ..	37·1
Monocytes	.. ..	7·6
Eosinophils	.. ..	3·2
Basophils	.. ..	0·2

Stained film showed some polychromasia.

The stitches were removed on Dec. 3, and the patient was discharged from hospital on Dec. 18, one month from the date of admission.

The capsule of the spleen on the convex side was raised by a subcapsular hæmatoma which had ruptured through the capsule in two different areas. Otherwise the spleen appeared normal in size and texture.

PATHOLOGICAL REPORT.—Mr. D. M. Greig sent the following report of the microscopical details : " Well-marked Malpighian bodies with central artery. Thickening and hyalinization of wall of central artery. No very distinctive pathological appearances."

Etiology.—The etiology and mechanism of rupture of the apparently normal spleen have been well discussed by Susman.[2] The spleen ages early, and at 36 years 50 per cent of spleens show thickening and hyaline changes in the Malpighian bodies and capsule. These degenerative changes tend to rupture. Congestion of the portal vein and its radicles will force blood between the spleen and its investing capsule of peritoneum, giving a sub-capsular hæmatoma. A slight degree of torsion of the splenic pedicle, which in a multiparous subject would be prone to twist, might cause the hæmatoma to burst.

SUMMARY.

1. A case of spontaneous rupture of an apparently normal spleen is recorded.

2. Only eleven authentic cases could be collected by Hamilton Bailey in January, 1930.

3. The etiology of rupture is problematical, but Mr. D. M. Greig, of the Royal College of Surgeons, Edinburgh, suggests in this case torsion of the splenic pedicle, which is feasible in a female of the maternal visceroptotic type such as the patient whose case is reported.

REFERENCES.

[1] BAILEY, HAMILTON, *Brit. Jour. Surg.*, 1929–30, xvii, 417.
[2] SUSMAN, M. P., *Ibid.*, 1927–28, xv, 47.

RESULT OF TRANSPLANTATION OF URETERS MORE THAN A QUARTER OF A CENTURY AFTER OPERATION.

BY P. R. ALLISON,

THE GENERAL INFIRMARY, LEEDS.

TRANSPLANTATION of both ureters into the rectum for the relief of ectopia vesicæ is an operation which has been performed on many occasions. Some of the cases succumb either as a result of the operation or from ascending pyelonephritis a few months or years later. The case reported below has survived for twenty-seven years in a state of good general health, and is therefore of sufficient rarity and interest to be reported.

The patient, E. V. B., male, aged 7 years, was admitted to the General Infirmary at Leeds on July 21, 1905, under the care of Mr. Lawford Knaggs, suffering from incontinence of urine since birth as a result of ectopia vesicæ. His mother picturesquely ascribed the deformity to a fall from a dog-cart which she sustained during pregnancy. When the child was 11 months old he was taken to the Infirmary, where Mr. Knaggs performed a plastic operation on the anterior abdominal wall; the boy, however, developed measles, and in consequence of his poor general condition the wound failed to heal.

During his stay in hospital he cried so much that the presence of a hitherto unsuspected left inguinal hernia became apparent. On discharge he was supplied with an instrument, consisting of a cage strapped to the abdomen with a tube leading into a receptacle on one leg, and with this he existed until the age of 7, but of this period of his life he remembers very little.

The accidental breakage of the instrument caused his mother to consult Mr. Knaggs again, with the result that the patient was readmitted to hospital and the operation of transplantation of ureters was undertaken. The following is an abstract of the clinical notes made at that time (1905) :—

On examination the upper two-thirds of the bladder are covered with thickened epithelium, whilst the lower part of the mucosa is raw and in a state of irritation. The urine can be seen dribbling from the ureteric orifices on to the raw area. The upper part of the penis is deficient, the testicles are not descended, and the pubic bones are separated by 2 to 3 in. The recti abdominis form a V-shaped hood to the bladder. A left inguinal hernia is present.

Operation was performed by Mr. Lawford Knaggs, on Aug. 1, 1905, under ether anæsthesia. The rectum was washed out. The bladder was first separated from its connections and from the peritoneum, leaving both ureters attached; the latter were splinted with catheters. Douglas's pouch was then separated from the rectum; the latter was pushed up by the fingers and a horizontal incision was made into it. The bladder was trimmed, leaving a portion with the ureters attached, which was drawn through into the rectum. The rectal aperture was closed by three sutures uniting the bowel wall and separating the ureters. The anal sphincter was dilated, a drainage tube inserted, and the suprapubic wound packed.

Following this operation the patient left Leeds and was not heard of for many years. Between the ages of 7 and 13 he went to school and played all

the usual games along with other children, but was, however, excused from the gymnasium. His general health was very good. About every two or three hours he passed per rectum a mixture of urine and fæces, and occasionally the intervals were as long as four hours. So long as he had the opportunity for evacuation at these times there was no incontinence, but if circumstances necessitated delay, there was considerable discomfort and even leakage. In addition to these liquid motions he passed a solid stool usually once a week.

From the age of 13 to 19 years he was lather boy and ran errands in a hairdresser's shop, after which he took a post as a lorry driver. This last occupation entailed the lifting of packages up to one and a half hundredweight and was indeed a very strenuous existence, yet he never felt any ill effects and was as competent and useful as his fellows. His day's work started at 7 a.m. and finished at 5 p.m., and at this time he was having a fluid evacuation every three to four hours and a solid motion every other day. In 1929 he married, but there have been no children.

His period of good health was only rarely interrupted by occasional colds, and in 1926 by an attack of lumbago which passed off in a few days. This consisted of an aching pain on both sides of the back, and was believed to have resulted from his heavy work : it was not associated with any pyrexia, sweating, or general ill health, so that it was probably not an acute renal infection. In 1930 he was off work for three days on account of a severe vomiting attack, but this appears to have been due to a dietetic upset and was not associated with any febrile symptoms or pain in the loins.

In July, 1931, he had his first serious set-back ; he began to feel ill, had cold shivers, vomited, sweated, and had a raised temperature. His doctor diagnosed influenza and kept him in bed for a fortnight. The patient subsequently complained of weakness, malaise, loss of appetite, and a numb pain in the left thigh, which was present only on standing and disappeared on lying down. In this condition he was admitted to the hospital under the care of Dr. Watson.

On admission on Aug. 13, 1931, he looked pale and appeared to have lost weight, his pulse was 88, temperature (in the axilla) 97·5°, and respiration 24. The tongue was clean.

The abdominal wall was well healed anteriorly, but was weak and required the support of a belt. Both testicles were in the scrotum and a left inguinal hernia was present. The penis was small and deficient on the dorsum, where at the junction with the abdominal wall there was a small raw area discharging a little mucous secretion. The pubic bones were separated by $2\frac{1}{2}$ in. There was tenderness and swelling in the left loin with some rigidity. The blood-count showed leucocytes 11,000 (neutrophil polymorphs 72 per cent, eosinophils 0·5 per cent, lymphocytes 21 per cent, monocytes 6 per cent). Blood-urea nitrogen was 26·1 mgrm. per cent.

On Aug. 17 the temperature was 99·6° and the pulse 96, after which there were frequent evening rises, on one occasion to 103°, with a pulse of 104.

An X-ray of the urinary tract showed a single calculus on the left side in a position corresponding to the pelvis of the kidney. Within a few days the swelling in the loin increased in size and became definitely fluctuant. On Aug. 25 the patient was transferred to the care of Mr. Flint, who incised the

loin and liberated a large quantity of foul-smelling pus. The abscess cavity was so large that its exact origin was difficult to determine. but. as neither stone nor kidney could be palpated, it was considered to be a large pyonephrosis.

On Aug. 20 a pyelogram was taken by means of intravenous injection of abrodil, and this showed a greatly hypertrophied kidney on the right side. The pelvis and calices were rather large, but the latter showed normal cupping. so that the increase in their size was presumably only in proportion to the degree of renal hypertrophy. No secretion of dye could be made out on the left side, which confirmed the suspicion that this kidney had been almost destroyed. Although the right ureter could be seen in parts the details of its structure and its opening into the rectum could not be determined. Sigmoidoscopy was not performed.

Following the operation the patient made rapid progress both in his general health and as regards the local condition. After discharging pus for six weeks the sinus in the loin almost healed except for a small quantity of serum, and on Sept. 30, 1931, he was discharged, apparently cured.

He says that when he returned home he felt so well that he wanted to go back to his work, but was advised to spend a few weeks breaking himself in gently. Four weeks later, however, he was quite suddenly taken ill and was readmitted to the hospital *in extremis*. At that time he was having repeated rigors and persistent hiccup ; the wound in the loin broke down and discharged urine and pus, and the blood-urea nitrogen was 36·6 mgrm. per cent.

On the advice of Mr. Flint a large tube was inserted into his rectum in order to drain the bowel ; urinary antiseptics and diuretics were administered. and on this treatment he gradually improved and was sent home to the care of his own doctor. From Dec. 1, 1931, until March, 1932, he remained in bed at home with the drainage tube in his rectum day and night, but by the latter date the sinus in the loin had healed completely, so he then discarded the tube during the daytime and started sitting up a little in the bedroom. In May, 1932, he had a femoral thrombosis in his left leg and had to return to bed. the leg being very swollen, cyanosed, and painful.

It was at this time that he discarded the rectal tube altogether, and the subsequent treatment has been directed against the condition of the leg, which still swells towards the end of the day. He is, however, up and walking about, and subjectively feels much fitter than he did during the two or three years preceding his recent illness. His appetite is very good indeed and he has gained weight so rapidly that a lumbar hernia has occurred through the loin incision ; this, however, is adequately controlled by a modification of the abdominal belt which he has always worn. At present he can retain his motions for four hours. and curiously enough he passes a solid stool every day. His blood-urea nitrogen on July 5, 1932, was 24·3 mgrm. per cent.

Another very interesting thing which is difficult to explain is that the patient can now tell whether the motion he is about to pass is a fluid or a solid one, and he was never able to do this before his illness. He does not pass flatus except along with his usual evacuations.

ENCYSTED PEDUNCULATED HÆMATOMA IN THE PLEURAL CAVITY IN ARTIFICIAL PNEUMOTHORAX.

By J. B. CHRISTOPHERSON,

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IT seems worth while to record the following account of a rare and interesting surgical and anatomical condition—namely, an encysted hæmatoma with a twisted pedicle in the pleural cavity of a male aged 23, whose lung was artifically collapsed for pulmonary tuberculosis. The hæmatoma evidently was due to an incident or accident. The intercostal vessels had in my opinion been wounded when unsuccessfully exploring to withdraw pleural fluid which
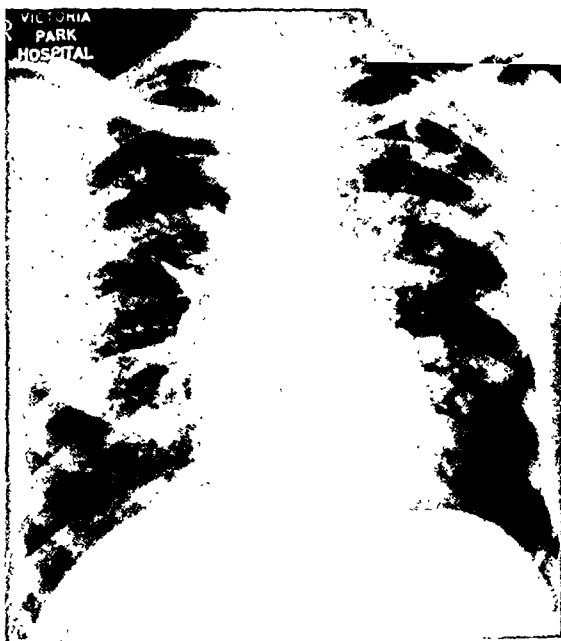
FIG. 317.—Jan. 13, 1932. Bilateral tuberculosis. Fair-sized cavity. Left apex seen below clavicle.

occurred in the course of artificial pneumothorax treatment. The hæmorrhage produced a subpleural tumour which, owing to the elasticity of the pleural membrane, became sausage-shaped, then, twisting on its long axis, acquired a pedicle and was a pedunculated cyst nine days later—with a twisted pedicle attached to the inner aspect of the thoracic wall. The accompanying skiagrams (*Fig.* 317–322) confirm my deductions. *Fig.* 317 was taken on the patient's admission to hospital on Jan. 13, 1932. It will be noted that these X-ray films are considerably clarified by the introduction

of lipiodol (*Fig.* 318, taken on May 15, about three weeks previous to the exploration on June 5 which caused the hæmatoma). -

In the film taken on June 11, six days after the date of the wounding of the intercostal vessels by the exploring needle, there is to be noted, in the anterior-posterior film, a small triangular shadow marked A (*Fig.* 319). Then if one turns to the lateral view taken on the same day (*Fig.* 320), there is to be noted a round shadow wholly submerged in the pleural effusion. This was evidently the cyst. which was not present in the film taken on May 15 (*Fig.* 318) about three weeks before the date of the exploration with

Fig 318.—May 15, 1932. Left artificial pneumothorax. Showing adhesions (3)—B and C to third and 4th ribs, another to dome ; pleural effusion ; lipiodol lies in pool unmixed with pleural fluid : some has adhered to thoracic wall and adhesions.

the needle and syringe. The four dots denoting the circular shadow were made by Dr. Sparks. Radiologist to Victoria Park Hospital.

On June 17 I did a thoracoscopy on the patient. dividing with the cautery three apical adhesions which were, I thought. causing the effusion ; after completing the division of the special adhesions I introduced into the thoracic cavity. through the thoracoscopic puncture. a catheter, and. with the excellent and efficient Chandler two-way syringe. withdrew 14 oz. of pleural fluid.

With the fluid withdrawn I obtained with the thoracoscope a clear view of the lower end of the thorax and saw lying on the side of the interior of the thorax. posteriorly. a bluish-white body resembling a half-inflated balloon. with a twisted pedicle lying on. and the pedicle attached to. the
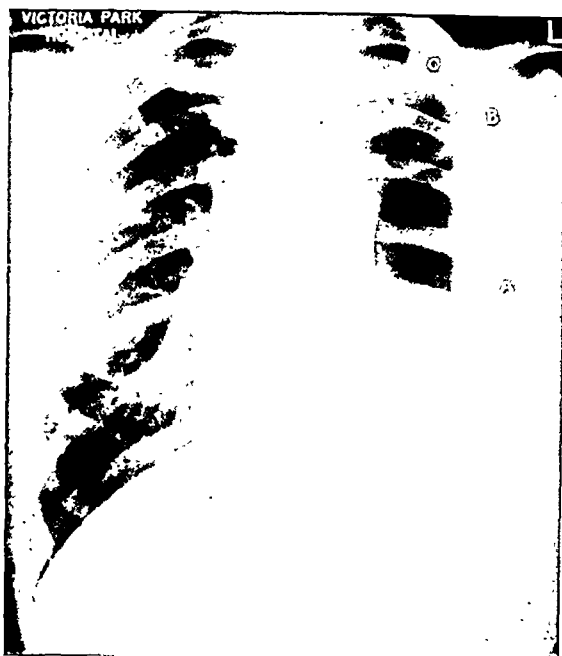
Fig. 319.—June 11, 1932. Skiagram taken after failure to evacuate fluid. Needle of exploring syringe was obstructed by cyst lying against posterior wall. A, Attachment of pedicle of cyst; B, C, Adhesions.

Fig. 320.—June 11, 1932. Lateral view. Loculated effusion. A, Base of stalk of adhesion. Globular mass attached to stalk and submerged. B, Lipiodol at bottom of fluid.

inner aspect of the thorax, I should judge tethered approximately where the needle had been used.

The cyst was evidently thin-walled, adapting itself to the shape of surrounding structures. This important point can be verified in the X-ray which I had taken immediately after the thoracoscopy (*Fig. 321*).

Finally, in the film (*Fig. 322*) made twelve days after the thoracoscopy operation (on June 29) the fluid was re-accumulating and the cyst had disappeared below the level of its surface, and so Miss Mickleburgh, the Radiographer at the City of London Hospital, Victoria Park, took the X-ray at about 5.0 p.m., the patient *lying on his side* in order to drain the fluid away from, and to isolate, the cyst. A very interesting film was obtained,
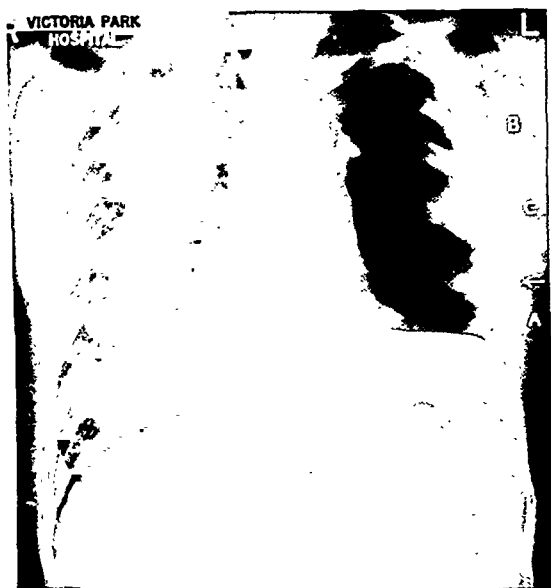
Fig. 321.—June 17, 1932. After thoracoscopy and division of adhesions by cautery. Soft purple tumour (D) with narrow long stalk attached to thoracic wall at A.   B, Lung stump after division of two adhesions.   C, Metal skin clip.

and attention is drawn to the now egg-shaped cyst, which had taken a brilliant coating of the lipiodol which I had introduced fourteen days previously (18 c.c.) for therapeutic reasons.

If this reproduction is viewed, as it was taken, in the horizontal position, a fluid level will be noticed situated above the diaphragm ; this is the pleural fluid displaced and lying over the mediastinum. Yet another fluid level is seen below the diaphragm : this is the level of the gastric contents—tea, etc., with the gastric air-bubble above—the man having just finished his tea at 5.0 o'clock before being X-rayed.

It may be added that there was no pain, no rise of temperature, and no other symptom complained of attributable to the cyst.

The case appears to point : (1) To the desirability of avoiding the inter-costal vessels by a knowledge of their relation to the ribs ; (2) To the use of lipiodol for enhancing radiological pictures of the pleural cavity ; (3) To the fact of the elasticity and extensibility of the pleural membrane ; (4) Incidentally, one might add to the fact that lipiodol is inert in the pleural
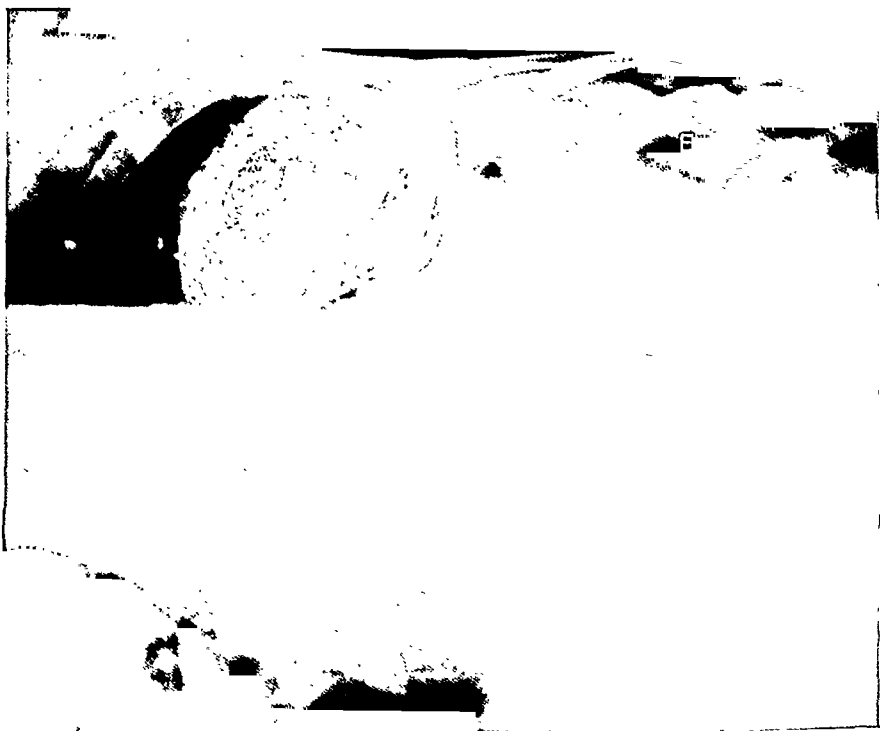
FIG. 322.—June 29, 1932. Skiagram taken with the patient lying on his side to displace fluid. Showing tumour (D) coated with lipiodol introduced on May 13 (18 c.c.). E, Gastric bubble.

cavity, that it takes a long time to absorb, that it does not mix with the pleural fluid, being an oleate, that it causes no inflammatory action in the pleura, or any action other than radiological—it is, in fact, therapeutically inert so far as the pleura is concerned.

Lipiodol is useful in ordinary radiology of the pleural cavity, also in stereoscopic radiology, and in thoracoscopy.

REVIEWS AND NOTICES OF BOOKS.

William Stewart Halsted. Surgeon. By Dr. W. G. MacCallum, with an Introduction by Dr. W. H. Welch. Demy 8vo. Pp. 241 + xvii. Illustrated. 1930. London : Oxford University Press. 12s. 6d. net.

The discovery of the practicability of general anæsthesia in the middle of the nineteenth century changed fundamentally the surgeon's outlook. Seemingly unlimited operative possibilities presented themselves to his mind. The rewards for daring and digital dexterity became tremendous. Dr. Halsted, in addition to being endowed with these qualities, possessed also the knowledge with which to implement them. Temptation lay before him. But for his fine, critical, and modest mind we might to-day remember him only as a figure that passed brilliantly through the operating-theatres of the eighties and nineties—a facile and ephemeral success. Instead, by guiding, and with personal discovery directing, the development of surgery he has made a permanent and unforgettable contribution to the welfare and happiness of human kind. In applying himself to the meticulously accurate surgery that strives continuously after a good physiological result he did much to protect the patient from the spectacular type of surgery, the disillusioning results of which are rapidly revealed by a study of the post-operative progress of its subjects. Dr. Halsted developed and taught a surgical technique as spectacular in its results as it was unspectacular in its performance.

But the founding of a great school of surgery expresses only a part of his activity. Every new and hitherto untried step taken in the operating-theatre was preceded, in so far as was possible, by minute and accurate clinical research upon animals in the laboratory. One of Dr. Halsted's happiest bequests to the students in Baltimore is the privilege they now enjoy of observing and taking part in this system of experimental study that he devised. In his early New York days he conceived ideas that were the basis of much fruitful research in that and later periods of his life. His block-dissection method in the treatment of cancer of the breast altered profoundly the prognosis in this affection. The operation he evolved for the cure of inguinal hernia has been of almost infinite benefit. So also have his discoveries in relation to local anæsthesia by the use of cocaine. His procedures in the treatment of aneurysm and in the treatment of acute infections of the gall-bladder are of established and recognized value. Dr. Halsted was one of the first surgeons in America to realize the enormous significance of Lister's discoveries. In face of the scepticism and unbelief of his colleagues he followed rigorously the antiseptic routine, and so, by dispelling the fear of the dreaded post-operative suppuration, completed the emancipation of the American surgeon that had been begun by the discovery of general anæsthesia. Not until Germany, and America under Halsted, had been for some time following the principles laid down by Lister did the English appreciate their value. Here, as always, Dr. Halsted was closely conversant with any surgical advance made in Germany, for whose surgeons he had a great and reciprocated respect.

This interest in German work and discovery dated from the two years Dr. Halsted spent in his youth as a post-graduate student in Germany, Austria, and France. During these years he made many friends. The high regard in which he held his foreign friends is proved by the generous way in which he helped to relieve the circumstances of those amongst them that were placed in difficulties after the war. His anxiety about the well-being of other surgeons exemplifies his concern for the well-being of surgery itself. One of Dr. Halsted's finest qualities was his

quick and unselfish appreciation of anything new and significant in the work of others.   In this way, by practising and interpreting the work of others, he performed some of his greatest services to surgery.   Pioneer, investigator, and distinguished teacher, he ranks with the great surgeons of his period.

History of Scottish Medicine.   By JOHN D. COMRIE, M.A., B.Sc., M.D., F.R.C.P., Physician to the Royal Infirmary, Edinburgh, etc.   Second edition.   Two volumes. Large 8vo.   Pp. 852, with 2 plates and 404 illustrations.   Published for the Wellcome Historical Medical Museum.   1932.   London : Baillière, Tindall & Cox.   50s. net.

THESE volumes, which form the latest addition to the Wellcome series of medical historical monographs, are of extreme value.   They are written by Dr. Comrie, who is Physician to the Royal Infirmary of Edinburgh and Lecturer on the History of Medicine in the Edinburgh University.   They have the merit of being exhaustive for the country with which they deal.   Scotland has long occupied a leading position both in the teaching and practice of medicine.   A succession of great teachers in the eighteenth century brought its schools into prominence and attracted students from England, from Ireland, and more especially from America.   Young men of talent who would have gone to Paris to learn anatomy and to Leyden for their clinical teaching now fared equally well at Edinburgh—or even better, for the language difficulties were less.   The foreign tour, which was universal in the seventeenth century, became less and less frequent without loss of the advantages which used to be gained from it.   The teaching of Boerhaave and Haller bore splendid fruit, for the earliest physicians like Archibald Pitcairne, Cullen, and James Gregory had been themselves taught at Leyden and Göttingen.   Midwifery, too, had always had a special attraction for the Scottish doctors because the midwives were untrained, very ignorant, and few in number.   The work of Smellie therefore appealed especially to them and the teaching was of a high order.   It was otherwise with surgery.   The country was poor, the means of transit bad, and the constant clan warfare made travelling dangerous.   Surgery, as we understand it, was only possible in Edinburgh and Glasgow, for even at Aberdeen there was no systematic teaching of the subject until late in the last century.   It was hampered, too, by its constant alliance with anatomy, and the Professor of Anatomy and Surgery was usually more interested in the dissections which he practiced daily than in the operations which he never undertook.   Clinical teaching was given as early as 1748 in medicine by those who had been taught at Leyden, and an attempt was made to hold classes in clinical surgery in 1769, but it was premature, and no regular classes were held until 1803.

Dr. Comrie gives some interesting accounts of the surgeons practising in Edinburgh.   He says " one of the best known of the eighteenth-century surgeons was Alexander Wood (1725–1807) known to his contemporaries as 'Lang Sandy Wood', and greatly respected for his dexterity in practice, which did much to raise the reputation of the surgical department in the Royal Infirmary, as well as beloved for his amiable social qualities."   He was represented by John Kay, who drew the well-known series of portraits of Edinburgh's worthies, in wig and cocked hat with an umbrella under his arm in allusion to the fact that he was the first person in Edinburgh to make use of that article.   At a time when personal peculiarity was widely affected in the capital Wood specially distinguished himself by going to see his patients accompanied by a pet sheep and a raven.   The life of a surgeon in Scotland was not usually cast in such pleasant places.   The customs of the period were primitive and curious.   A surgeon would start on Monday caparisoned for the week with drugs and surgical appliances and would not return home until Friday.   To obviate the dangers of travelling by night he carried a lantern fastened by a strap above his knee.   The bull's-eye of the doctor's lantern was often signalled on moonless nights, heralding the comforting assurance of an obstetric deliverance. His regularity in his rounds vied with the carrier of His Majesty's mails; the saddle-bags of the one and the surgical accoutrements of the other were similarly horsed so that the laird scanning the road could say, " It's either the doctor or the post that's

coming." By such sidelights Dr. Comrie lightens the history and makes it most readable. He has, too, been at great pains to select lifelike portraits of the men he mentions, and the whole book is illustrated lavishly. Chapter and verse are given for all the statements, so that it is easy to test their accuracy and obtain more detailed information. There is a good index and the book is excellent from beginning to end.

Chirurgische und konservative Kosmetic des Gesichtes. Edited by Dr. LEANDER POHL (Vienna). Large 8vo. Pp. 383 + viii, with 445 illustrations and 3 coloured plates. 1931. Berlin and Vienna : Urban & Schwarzenberg. Paper covers, RM. 25 ; bound, RM. 28.

THIS book will appeal to few practitioners in this country, for as yet no specialty entirely devoted to cosmetic medicine has appeared in England. Nevertheless, that the subject is considered of growing importance on the Continent is evidenced by the recent increase in German and French literature devoted to it, and by the fact that this latest addition edited by Dr. Pohl is issued under the ægis of Professor von Eiselsberg. It represents a collaborative attempt to group together all those medical and surgical affections of the face which may be described as of cosmetic importance. Thus it invades boldly fields which in this country have hitherto belonged almost exclusively to the physician, the dermatologist, the roentgenologist, the dentist, the general surgeon, and finally to the already highly specialized plastic surgeon.

It contains a great deal of interesting material and much that can be obtained only from scattered sources, and for that reason is a valuable contribution. The various sections, however, suffer from a certain unevenness both in quality and completeness which rather detracts from the usefulness of the book as a whole.

Pohl contributes an interesting study of facial proportions and their variations from youth to old age. A complex analysis of facial components and their combinations both in profile and full face enables him to separate individuals into definite facial groups. For this purpose he uses a measuring instrument and a photographic recording device. Facial appearance characteristic of a wide variety of general diseases is discussed by Kollert, while the more local affections of the face are dealt with by Stein. Holzknecht contributes an account of his treatment of hypertrichosis by X rays, a method no longer in vogue in England. The plastic surgical treatment of defects of the cheeks, nose, mouth, ears, and eyelids is dealt with by Frisch, Safar, and Ehrenfeld. This section follows conventional lines and might have been materially improved by better diagrams and illustrations. Plastic surgery is dominated largely by mechanical difficulties and lends itself particularly well to graphic and pictorial illustration. Pichler deals with lesions of the lower jaw requiring resection, bone-grafting, or the use of prosthetic appliances. The section devoted to micrognathism and macrognathism is of considerable interest.

The chapter on cleft lip and palate is rather disappointing. Neither Brophy's nor Mirault's operation warrants serious consideration in an up-to-date discussion of this subject. No mention is made of the excellent contributions of Vaux, Ritchie, or Davis, nor of the opinions so often expressed by Gillies, Fry, and Wardill on the necessity and means for obtaining apposition between the soft palate and the posterior pharyngeal wall for functional success. Another omission is the treatment of secondary defects of the lip following primary closure—a matter of paramount cosmetic importance and a type of defect frequently encountered in the practice of plastic surgeons. The book concludes with a section devoted to malformations, malpositions, and diseases of the teeth, and their treatment by medical and orthodontic means.

The volume covers so wide a range of subjects that one cannot escape the feeling that the field is too comprehensive ever to be considered as one specialty or to be efficiently handled by one person. On the other hand, it indicates the supreme importance of close co-operation between medical, surgical, and dental experts in the cosmetic treatment of facial affections.

The Acute Abdomen. By C. H. FAGGE, M.S. Lond., F.R.C.S., Surgeon to Guy's Hospital. *Pocket Monographs on Practical Medicine.* F'cap 8vo. Pp. 92 + viii. 1932. London : John Bale, Sons & Danielsson, Ltd. 2s. 6d. net.

THIS volume is one of the Pocket Monographs on Practical Medicine, and is a very small book dealing with a large and difficult subject. Mr. Fagge, however, manages to give the reader a wealth of information based on his extensive practical experience. The book is divided into three parts. In the first the various symptoms met in an acute abdominal catastrophe are outlined and their significance and varying importance discussed. The next part considers the differential diagnosis of the individual lesions, and the final part the principles of treatment. On debatable subjects, such as the treatment of acute appendicitis, Mr. Fagge states his views from the aspect of pathology and practical experience. We cannot agree, however, that the point of tenderness in acute appendicitis does not alter with the different positions of the appendix.

The whole of the matter is set out in a perfectly clear and simple manner, and the book can be read in a few hours under armchair conditions. There are no illustrations. House surgeons and many others will benefit by a perusal of this volume, and they will not go far wrong if they adhere to the author's advice but at the same time understand the basis of his reasoning.

A Clinical Study of the Abdominal Cavity and Peritoneum. By EDWARD M. LIVINGSTONE, B.Sc., M.D., Associate Visiting Surgeon, Bellevue Hospital, New York. Imperial 8vo. Pp. 866 + xxii, with 372 illustrations. 1932. New York : Paul B. Hoeber, Inc. $15.00.

IN this book the author has compiled a comprehensive review of abdominal symptoms and signs with special reference to the embryological, anatomical, and physiological factors concerned. The work is intended for students or graduates, and by its arrangement can easily be used to a good purpose by either.

Of the many sections, those dealing with the nervous mechanism of the abdomen —both motor, sensory, and sympathetic—are perhaps the most striking; these chapters are freely illustrated with useful and informative charts and diagrams. In the final chapter on the clinical interpretation of viscero-sensory phenomena a full and careful estimation is given of the value of the various sensory skin manifestations in intra-abdominal affections. As a work of reference the book will undoubtedly serve a useful purpose.

Diagnosis in Joint Disease: A Clinical and Pathological Study of Arthritis. By NATHANIEL ALLISON, M.D., F.A.C.S., Professor of Surgery, University of Chicago, etc.; and RALPH K. GHORMLEY, M.D., Associate in Orthopedic Surgery, Mayo Clinic, etc. Large 4to. Pp. 196 + xii. Illustrated in colour and in black-and-white. 1931. London : Humphrey Milford. 52s. 6d. net.

THIS monograph deals with all the principal forms of arthritis—tuberculous, traumatic, loose bodies, pyogenic, rheumatoid, and osteo-arthritis. In each section there are many clinical histories of typical cases, radiographs of early, moderate, and late stages of the disease, and histological pictures of the tissues involved. This method of presentation gives a very clear and complete account of each disease, and the accuracy and beauty of the illustrations call for great praise. The only criticism of the illustrations which we can make is that the reproductions of colour photographs are disappointing, because they give no clear definition of the conditions depicted. This, however, only applies to the frontispiece and to *Plate VII.* The terms 'rheumatoid arthritis' and 'osteo-arthritis' are avoided, the former being chiefly covered by the phrase 'proliferative arthritis', and the latter by that of 'degenerative arthritis', but in each case the illustrative figures give an excellent picture both of the joints and the tissues which are affected.

The book as a whole will form an admirable source of reference.

Minor Surgery of the Urinary Tract. By H. C. BUMPUS, Jun., with chapters on Caruncles by J. L. CRENSHAW, and on Postoperative Care by A. L. CLARK. 8vo. Pp. 124, with 57 illustrations. 1932. Philadelphia and London : W. B. Saunders Company. 15s. net.

THIS book is one of the Mayo Clinic monographs, and gives in a clear and concise manner descriptions of minor surgical procedures as practised in the genito-urinary department of that institution. It opens with a short account of general considerations such as anæsthesia, preparation of the patient, etc. Next are given the details of the neat and effective method of dealing with that painful lesion, the urethral caruncle. Perhaps the most interesting chapter is that concerned with hypertrophy of the prostate gland. Bumpus here describes his modification of the Braasch 'punch' operation for the removal of the obstructing portion of the gland. This endoscopic method of treatment of the enlarged prostate may now be said to have passed from infancy to adolescence, and in the future is obviously destined to take an increasingly prominent place in the surgery of this condition.

Two interesting and rare affections of the bladder—namely, interstitial cystitis (Hunner's ulcer) and alkaline phosphatic cystitis—are discussed in considerable detail. The manipulative extraction of stones in the ureter is a subject which is treated with thoroughness, the several methods of treatment employed being described fully. With most of the post-operative care suggested we are familiar, but an unusual feature of this chapter is that on the use of a ketogenic diet as a means of acidifying the urine. We imagine that this method of altering the pH of the urine, as an adjuvant in the treatment of infections of the urinary tract, is still in the experimental stage. However, if it can be proved definitely to be of use in cases of chronic pyelonephritis, then it is surely worthy of consideration.

Amongst surgeons in general, and in particular amongst those who are interested in surgery of the urinary tract, this small monograph is likely to prove deservedly popular.

Diseases of the Kidney. By W. GIRLING BALL, F.R.C.S., Surgeon to St. Bartholomew's Hospital ; and GEOFFREY EVANS, M.D. (Cantab.), F.R.C.P. (Lond.), Physician, with charge of Out-patients, St. Bartholomew's Hospital. Royal 8vo. Pp. 424 + viii, with 159 illustrations and 8 coloured plates. 1932. London : J. & A. Churchill. 36s. net.

SINCE Rayer published his omniscient book, *Les Maladies des Reins,* the subject has become so vast and complicated that there must be few indeed who would venture to write a monograph which should bring up to date our knowledge of the diseases of the kidney from both the medical and surgical points of view. This volume is a noteworthy attempt to do so within the limits of a book of reasonable size ; the authors are a surgeon and a physician at St. Bartholomew's Hospital, and, with the wonderful traditions of that ancient hospital to inspire them, we expect a monograph which will speedily become a classic.

The book is well bound and beautifully printed, and the numerous illustrations are delightful and a real help in the elucidation of the text ; the latter, as one would expect of these two authors, is plain and straightforward, and most singularly free from printer's errors.

The chapters on movable kidney and tuberculosis of the kidney struck us as particularly good ; under the former, we are relieved to find that the authors do not look on the occasional fixation of a movable kidney as a surgical misdemeanour, and their account of tuberculous disease of this organ is one of the best we have read.

There are one or two rather surprising omissions : we can find no mention of either hydatid disease or of actinomycosis, though both are well recognized, if very uncommon, affections of the kidney. The description of oxaluria would have been more complete if the question of the excessive flatulence which sometimes accompanies the attacks had been discussed. This omission is the more glaring since the best account of this syndrome was published by one of the authors' colleagues in the *Transactions* of the Medical Society of London. He gave an account of a house surgeon whose intestinal symptoms were so marked that a

laparotomy for intestinal obstruction was done; his condition after this operation was unchanged, and a second operation was contemplated, but, at a consultation of the Staff, it was found that the first examination of the urine had been wrongly reported and that it contained a good deal of blood and was full of oxalates.

Bright's disease is very thoroughly discussed, a chapter of nearly one hundred pages being devoted to it; we have read it through carefully, and our conclusion, owing perhaps to surgical prejudice, is that this disease has become so complicated as to be almost incomprehensible.

These are, however, very minor defects, and we can most heartily commend the book to our readers.

Intracranial Pyogenic Diseases. A Pathological and Clinical Study of the Pathways of Infection from the Face, the Nasal and Paranasal Air-cavities. By A. Logan Turner, M.D., LL.D. Edin., Hon. F.R.C.P.E., F.R.S.E., F.R.C.S.E., Consulting Surgeon, Ear and Throat Department, Royal Infirmary of Edinburgh; and F. Esmond Reynolds, M.D. Edin., D.T.M.&.H. Camb., M.R.C.P.E., Lecturer on Neuropathology, University of Edinburgh. Royal 8vo. Pp. 271 + xx, with 82 illustrations. 1931. Edinburgh: Oliver & Boyd. 12s. 6d. net.

This volume, inscribed as it is " to the pioneer work of Sir William Macewen on intracranial infection ", constitutes a fitting companion to that great surgeon's well-known classic, published in 1893, under the title of *Pyogenic Diseases of the Brain and Spinal Cord*. It embodies the results of some ten years' methodical and painstaking research into the pathways of intracranial infection, correlating the clinical and pathological observations in a series of 55 cases. In most of these the primary septic lesion was connected with the nasal and paranasal air cavities. In passing it may be remarked that the authors have rendered a service to clearness of nomenclature by rejecting the term 'sinus' as applied to the air-spaces in the bones. Throughout the book they designate these as 'air cavities', and reserve the word 'sinus' for describing the venous intradural spaces.

Of the 55 cases, the primary intracranial complications were leptomeningitis in 24, cavernous sinus thrombosis in 21, and cerebral abscess in 4. The remaining 6 cases were examples of generalized hæmic infection or pyæmia.

The beginnings of cerebral abscess in the authors' cases are clearly shown to have been retrograde thrombosis of veins followed by infection of the perivascular spaces.

Septic thrombosis of the cavernous sinuses occupies a large part of the book. These cases are divided into 'fulminating' (5), 'acute' (3), and 'restrained' (13). The mechanism of the ocular symptoms is fully discussed. Of the 21 patients, 1 recovered. In this case the primary focus was aural disease on the left side, with unilateral cavernous sinus thrombosis on the right side. There seems to be little ground for believing that in this instance the thrombosis was of a septic nature.

The book is excellently produced and well illustrated, 21 of the 82 pictures being beautifully coloured.

The Use of Lipiodol in Diagnosis and Treatment. By J. A. Sicard, Late Professor in the Faculty of Medicine, Paris, and J. Forestier (Aix-les-Bains). Royal 8vo. Pp. 235 + x, with 50 illustrations. 1932. London: Humphrey Milford, Oxford University Press. 16s. net.

The French edition of this work appeared in 1928. It deals chiefly with the diagnostic applications of lipiodol, and the various methods of its use are given in great detail. Possibly its utility in the localization of spinal tumours and other causes of spinal compression is overestimated, whilst the objections to its use in these cases are, though frankly discussed, minimized. Little, for instance, is made of the root pains and sphincter disturbances which sometimes follow intrathecal injection. Possibly such serious ill effects have been due to the use of lipiodol which has undergone decomposition. The authors insist strongly against the use of lipiodol which no longer possesses its original pale amber colour.

The account of the therapeutic uses of lipiodol occupies but fifteen pages. It has been employed for pain in various positions and of varying causation; and also for such conditions as chronic arthritis, tuberculous abscess, and bronchiectasis. The therapeutic effects do not appear to be such as to arouse enthusiasm.

It is rather surprising that of the 231 pages, 39 are devoted to bibliography, and that these pages contain references to no fewer than 708 papers relating to the subject.

The reproductions of the radiograms are greatly superior to those in the French edition.

St. Bartholomew's Hospital Reports. Edited by Sir THOMAS HORDER, Bart., K.C.V.O., RONALD G. CANTI, WILFRED SHAW, CHARLES F. HARRIS, J. PATERSON ROSS, R. C. ELMSLIE, W. GIRLING BALL, and GEOFFREY EVANS. Vol. LXIV. Demy 8vo. Pp. 231 + xxv. Illustrated. 1931. London: John Murray. 21s. net.

WHEN reviewing recent volumes of this series, we have had occasion to dwell favourably upon the peculiar happy notes which have appeared over the initials of present members of the staff in commemoration of the careers of their old teachers. Dr. Barris's obituary notice of Sir Francis Champneys is in no particular below the customary high level. It draws attention to Champneys' many-sidedness without unduly obscuring his prominence as a gynæcologist. It is sad to reflect how often one fails to glean the interest of one's acquaintances until their obituary notices appear. It may be news to many to read of Champneys' reputation as a learned and admirable musician.

Professor H. H. Woollard's paper on spastic paralysis contains a clear and up-to-date account of work on the physiology of the sympathetic system and the way in which it has been applied to the surgery of spastic paralysis.

J. P. Hosford writes very briefly on the treatment of common fractures. It is to be hoped that in a later volume he will write in more detail of his experience in the use of Kirschner's wire for axial traction.

Ralph Phillips contributes a very long and painstaking study of buccal cancer, which was a thesis approved for the degree of Master of Surgery in the University of London. Perhaps his chief conclusion is a corroboration of the common belief that the histological appearance bears a well-marked correlation with the clinical course of cancer.

BOOK NOTICES.

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

Rose Research in Lymphadenoma. By Sir THOMAS HORDER. Bart., K.C.V.O., M.D., F.R.C.P.; M. H. GORDON, C.M.G., C.B.E., M.A., D.M., F.R.S.; KENNETH STONE, D.M., M.R.C.P.; LAWRENCE P. GARROD, M.A., M.B., M.R.C.P.; E. R. CULLINAN, M.D., B.S., M.R.C.P.; and B. D. PULLINGER, M.D., B.S. Crown 4to. Pp. 136 + viii, with 98 illustrations in colour and in black-and-white. 1932. Bristol: John Wright & Sons Ltd. 21s. net.

The Science and Practice of Surgery. By W. H. C. ROMANIS, M.A., M.B., M.Ch. (Cantab.), F.R.C.S. (Eng.), F.R.S. (Edin.), Surgeon and Lecturer on Surgery, St. Thomas's Hospital, etc.; and PHILIP H. MITCHINER, M.D., M.S. (Lond.), F.R.C.S. (Eng.), Hon. Surgeon to H.M. the King, etc. Fourth edition. Royal 8vo. In two volumes. Vol. I, General Surgery. Pp. 865 — x, with 373 illustrations. Vol. II. Regional Surgery. Pp. 1045 — x, with 326 illustrations. 1932. London: J. & A. Churchill. 14s. net per vol.

Treatment of Fractures in General Practice. By W. H. OGILVIE, M.D., M.Ch., F.R.C.S. F'cap 8vo. Vol. I, Pp. 108 — viii; Vol. II, 109–180. Illustrated. 1932. London: John Bale, Sons & Danielsson Ltd. 2s. 6d. each.

The Relative Value of Radiotherapy in the Treatment of Cancers of the Upper Air-passages. University of London Semon Lecture. By W. Douglas Harmer, M.A., M.B., M.C. (Cantab.), F.R.C.S. Demy 8vo. Pp. 85 + vi. Illustrated. 1932, London : John Murray. 6s. net.

Acromegaly. By F. R. B. Atkinson, M.D., C.M. (Edin.), with a Foreword by Sir Arthur Keith. Demy 8vo. Pp. 260 + x, with 3 plates. 1932. London : John Bale, Son. & Danielsson Ltd. 21s. net.

Kleine Chirurgie. By Prof. Dr. Hans Kurtzahn (Königsberg i. Pr.). Second edition. Large 8vo. Pp. 462 + viii, with 167 illustrations. 1932. Berlin and Vienna : Urban & Schwarzenberg. Paper covers, RM. 13.50 ; bound, RM. 15.

St. Bartholomew's Hospital Reports. Edited by Sir Thomas Horder, Bart., K.C.V.O., Ronald G. Canti, Wilfred Shaw, Charles F. Harris, J. Paterson Ross, R. C. Elmslie, W. Girling Ball, and Geoffrey Evans. Vol. LXV. Large 8vo. Pp. 1–317, with Index of Vols. XLI–XLV. Illustrated. 1932. London : John Murray. 21s. net.

A Synopsis of Surgical Anatomy. By Alexander Lee McGregor, M.Ch. (Edin.), F.R.C.S. (Eng.), Lecturer on Surgical Anatomy, University of Witwatersrand, etc., with a Foreword by Sir Harold J. Stiles, K.B.E., F.R.C.S. (Edin.). Crown 8vo. Pp. 609 + xvi, with 606 illustrations. 1932. Bristol : John Wright & Sons Ltd. 17s. 6d. net.

Erdmann's Clinics. Excerpts selected from the Clinics of John F. Erdmann, M.D., F.A.C.S., Professor of Surgery in Colombia University. Edited by J. William Hinton, M.D., F.A.C.S., Associate Professor of Surgery, New York Postgraduate Medical School. Large 8vo. Pp. 315 + vi, with 39 illustrations. 1932. Philadelphia and London : W. B. Saunders Company. 22s. 6d. net.

Practical Anatomy by Six Teachers. Edited by E. P. Stibbe, F.R.C.S., Senior Demonstrator in Anatomy, London Hospital Medical School. Demy 8vo. Pp. 719 + xii, with 337 illustrations. 1932. London : Edward Arnold & Co. 30s. net.

A System of Surgery. Edited by C. C. Choyce, C.M.G., C.B.E., B.Sc., M.D., F.R.C.S., Professor of Surgery in the University of London, etc. Pathological Editor, J. Martin Beattie, M.A., M.D., C.M., M.R.C.S., Professor of Bacteriology in the University of Liverpool, etc. Third edition. In three volumes. Medium 8vo. Vol. I, pp. 1112 + xxiv, with 33 colour and 67 half-tone plates, and 285 illustrations in the text. Vol. II, pp. 1111 + xvi, with 16 colour and 11 half-tone plates and 367 illustrations in the text. Vol. III, pp. 1107 + xvi, with 11 colour and 39 half-tone plates and 277 illustrations in the text. 1932. London : Cassell & Co. Ltd. £6 net the three volumes.

A Shorter Orthopædic Surgery. By R. Brooke, M.S., F.R.C.S., Hon. Orthopædic Surgeon, Royal West Sussex Hospital. Large 8vo. Pp. 150, with 126 illustrations. 1932. Bristol : John Wright & Sons Ltd. 10s. 6d. net.

The Injection Treatment of Varicose Veins, Hæmorrhoids and Other Conditions. By Rodney H. Maingot, F.R.C.S., Surgeon, Royal Waterloo Hospital, London, etc. Crown 8vo. Pp. 100 + xii. 1932. London : H. K. Lewis & Co. Ltd.

The Early Diagnosis of the Acute Abdomen. By Zachary Cope, B.A., M.D., M.S. (Lond.), F.R.C.S., Surgeon, St. Mary's Hospital Paddington, etc. Sixth edition. Large 8vo. Pp. 248 + xiv, with 30 illustrations. 1932. London : Humphrey Milford, Oxford University Press. 10s. 6d. net.

The Principles and Practice of Rectal Surgery. By William B. Gabriel, M.S. Lond., F.R.C.S., Surgeon to St. Mark's Hospital and the Royal Northern Hospital. Royal 8vo. Pp. 248 + viii, with 118 illustrations, including 8 coloured plates. 1932. London : H. K. Lewis & Co. Ltd. 20s. net.

Reinterventi operativi. Patologia e clinica. By Dott. Giovanni Razzaboni (Parma). Large 8vo. Pp. 935, with 626 illustrations. 1932. Bologna : Licinio Cappelli.

Classical Descriptions of Disease. With Biographical Sketches of the Authors. By Ralph H. Major, Professor of Medicine, University of Kansas Medical School. Large 8vo. Pp. 630 + xxviii, with 127 illustrations. 1932. London : Baillière, Tindall & Cox. 30s. net.

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SIR ROBERT JONES, Bart., K.B.E., C.B.

ROBERT JONES is dead. England parts in grief from one of her greatest surgeons and finest gentlemen; orthopædic surgery loses the supreme master.

Robert Jones was born in 1858, qualified as M.R.C.S. twenty years later, and began practice in association with his uncle on the maternal side, Hugh Owen Thomas, one of the most original minds in surgery. Thomas was the son of a famous Liverpool bone-setter, who inherited a practice that had descended through seven generations from father to son. The partnership which existed between Thomas and his father, an unqualified practitioner, was severed by the Medical Act of 1858. Robert Jones was fortunate in this association. His uncle, to whose memory he was devoted, made him his disciple, transmitted to him a legacy of all the craftmanship the most expert of bone-setters had acquired, and kept his mind at work by the novel, even new-fangled, views and methods of which he never ceased to speak.

The Thomas's splints and wrench are only a small part of the inheritance bequeathed to surgery by this strange, eager, unconventional surgeon. The world at large took many years to recognize their value, for although the practice of Thomas, and later of Jones, was numerically the largest in the kingdom, few beyond the area of Lancashire, West Yorkshire, Cheshire, and North Wales had realized that two of the most remarkably gifted men were unceasingly at work, and that between them they had almost silently raised orthopædic surgery to a level incomparably higher than that reached in any other part of the world. It was not until a little over thirty years ago that we began to hear much of Robert Jones. American surgeons, beginning with the Mayos, used to visit Liverpool, Edinburgh, and Leeds, to see the work of Robert Jones, Harold Stiles, and Moynihan. The three heard much of each other, took opportunities to meet, with the result that, in order to afford easy occasions for like friendships among other provincial surgeons, a club was founded, which, meeting twice yearly, once at home, once abroad, soon created new friendships, increased intellectual traffic among its members, improved the technique and developed the skill of every surgeon engaged in teaching in provincial schools.

- SIR ROBT. JONES, BART., K.B.E., C.B.

life devotion to their task done as he would wish it to be done can repay.

It was a wonderful experience to visit Robert Jones and to see him doing his routine hospital work. On arrival one was presented with a list of twenty-five operations to be performed ! Robert Jones worked with an almost incredible speed, and was supported by a team as efficiently trained as any one has ever seen. There seemed a silent conspiracy of united relevant effort in which every single person from surgeon to theatre porter took part. The surgical technique was flawless : there was none better in the whole world, and Jones enjoyed the distinction of being a fast operator whose technical methods had no weakness and did not give way under the strain of speed. There is far too often a sacrifice of safety to haste ; speed should be the accomplishment, not the aim. of an operator. As Jones worked. the clock appeared to be losing time. There was no slightest suspicion of hurry. One act followed another in due order without fuss. without delay ; before one was prepared for it the list of operations was completed, and one began to wonder whether anything could conceivably go wrong with such superb unapproached mastery of diagnosis, of perfection in technique, of infinite resource. Many of Robert Jones's methods were of his own devising ; he would employ, almost without one being quick enough to notice it, a device which alone would have made the reputation of another man. He would fashion a splint, and make one feel that the pliant metal knew what was expected of it. He was. in fact, a consummate artist. in design and in accomplishment.

It is impossible to tell truly of the love his patients bore him. He was greeted by the children in the open-air wards at Heswall. not with quiet respect. but literally with a yell of delight. His manner to them all. his patience, tenderness, loving care, his sweetness and infinite gentleness, were a lesson to all. It may be doubted if any man ever won such love from patients as he did ; his charm and kindness and compassion were infinite.

Robert Jones was a man of great physical power. Bones lost their strength when he bent or broke them. Twisted feet became supple, and as he moulded them took on a new and better shape. And he was above the tyranny of fatigue. He went from one task to another with the gay and inexhaustible sprightliness of youth, and wherever he went men's hearts were lightened, their courage strengthened and their joy increased, their ideals made more glowing.

On his seventieth birthday he was presented with a *Birthday Book* composed of original scientific essays, chiefly on orthopædic subjects, by twenty-five surgeons. his friends, pupils, and co-workers. These included eight London. five foreign, and three colonial surgeons. The book was presented to him at a special dinner, and the words of its preface, by his life-long friend Lord Moynihan, serve as a fitting epilogue to this article :—

Spirit alone is immortal. In the practice of orthopædic surgery the spirit of Robert Jones will live for ever.

The story of the triumphs of Robert Jones as prophet, high priest, and practitioner in orthopædic surgery makes plain the reason for our deep respect. Our affection for him transcends, if it be possible, our gratitude for his professional

worthiness. Few man have ever possessed in so radiant a degree the genius for friendship. No one can be long in his company, none can work with him or play with him, without realizing not only the sweet simplicity of his character, but the greatness of his heart. He speaks ill of no man. He seeks and finds good in all things and in all men. He sets an ideal and a standard of action in friendship which all strive to reach when with him. In a long and very intimate friendship I have never heard an unjust criticism, a cruel jibe, or a word of bitter cynicism on his lips. He covers his displeasure or stern disapproval by silence, or a restrained reproof that is often weightier than a torrent of words from others. His personality radiates cheeriness, good temper, and good will. All men are attracted by him, and in war-time conflicting temperaments found in him something that appeased their differences, assuaged animosities, and encouraged a desire for friendliness. He was then called upon to rule in various places, over colleagues at first unfriendly, openly antagonistic, indifferent to his rule, or incredulous of his practice. We were all amazed at his success in overcoming very real difficulties by gentleness, sympathy, a true understanding of the minds of others, and a tactfulness which in times of crisis was almost magical.

It is the simple truth to say that within the memory of the present generation no man in our profession has been so deeply, so universally, beloved. He was the perfect friend.

SIR PERCY SARGENT, c.m.g., d.s.o.

SIR PERCY SARGENT, C.M.G., D.S.O.

PERCY SARGENT was born in 1873 and educated at Clifton College. From Cambridge he went to St. Thomas's, where he won the University Scholarship in 1895. He was House Surgeon in 1899 and Resident Assistant Surgeon in 1903. To his seniors at the hospital Sargent's talents became very obvious while he was House Surgeon. Even at this date he showed that skill and dexterity for which he subsequently became famous; he also showed an independence of thought that was usually tempered with a respect for the opinion of others. While holding the post of Resident Assistant Surgeon the qualities he displayed made it certain that he would become a member of the staff, and to that position he succeeded in 1905.

A little later Queen Square were looking about for a Junior Surgeon, and Sargent was chosen to carry on the tradition of Horsley. To this end he applied himself with vigour and enthusiasm. It is uncertain whether the choice of cerebral surgery arose spontaneously or whether the chance of a post at the National Hospital turned his thoughts in this direction; possibly the association with Ballance influenced his choice. Be that as it may, the Great War found him with a developed technique made up of rapidity, gentleness, and precision, which contrasted somewhat with that which is practised in some quarters to-day.

With Gordon Holmes he formed a neurological unit which led towards a better knowledge of localization of functions in some parts of the brain. He returned to London before the end of the war and took charge of a department for those still suffering from remote injuries of the nervous system, and eventually became Consulting Surgeon to the Ministry of Pensions.

He played a large part in the reorganization of his School which was necessitated by the disturbances caused by the war, and here his alertness and enthusiasm found a further outlet.

Although Sargent chose a special line of work, he never let it push general surgery into the background, and he continued to the end as interested in general surgical problems as any of his colleagues. He had a good judgement and was never the heroic surgeon in spite of his quickness and dexterity. He never strove after advertisement by publication, and that which he wrote was clear and to the point.

He gave the Erasmus Wilson Lecture at the College of Surgeons in 1905, was elected to the Council of that College in 1923, and was Vice-President at the time of his death. Ever since 1923 he has been a most active member of the Editorial Committee of this JOURNAL, and his kindly help to younger men in the presentation of papers as well as his critical judgement in the conduct of the JOURNAL will be difficult to replace.

Sargent was at most times very receptive and keen to try new methods and new technique, but he never adopted permanently any mechanical aids to operation; this was largely due to his manual dexterity.

He was a good colleague, intensely interested in and loyal to his School; this attitude appeared to make him at times a little blind to its imperfections and the perfections of others; perhaps this blindness was intentional. Sargent had sound friends; he also had that valuable gift of getting on with strangers. He was a cheerful companion with a rather piquant and trenchant way of putting things, but somehow the things he said never rankled, and this was true not only of his private conversation but of his performances on public social occasions, when he could be daring.

He was never a robust man, and a physical disability told heavily on him at times, though it was hidden from view. Perhaps it was this acquaintance with periods of ill health that made him interest himself in those on whom misfortune had fallen.

THE TREATMENT OF TUBERCULOUS DISEASE OF THE HIP-JOINT.*

By A. S. BLUNDELL BANKART,

ORTHOPÆDIC SURGEON. MIDDLESEX HOSPITAL, ETC.

DURING the last twenty-five years we have seen the growth and development of a most elaborate and costly system of treatment for surgical tuberculosis in general, and for tuberculous disease of the hip in particular. Large special hospitals have been provided in many places for the prolonged constitutional and local treatment of this disease. At these hospitals open air, sunshine, artificial light in some cases, special diet, education, and amusement have been combined with the most careful and painstaking conservative treatment of tuberculous joints for periods varying in different cases from one to five years. Yet, in spite of all this, some of us are profoundly dissatisfied with the results of the treatment of tuberculous hip. It is true that from some quarters we hear of wonderful results with high percentages of cures and perfect restoration of function ; but this is not the experience of most of us, and frankly we are sceptical.

Actually we see case after case of active tuberculous disease of the hip which becomes quiescent or apparently cured under modern conservative treatment, and then after a longer or shorter interval—it may be years—it becomes active again and requires further treatment. It is abundantly evident that in such cases the disease has never been cured but has been latent and a constant source of danger to the patients in later life. What the percentage of these cases is, we do not know ; but it is so considerable that some people doubt whether tuberculous disease of the hip-joint is ever cured by conservative treatment. I must confess that after a long struggle against what I thought was my better judgement, I have come to much the same conclusion.

Great stress has been laid upon the fact that tuberculous arthritis is a metastatic infection secondary to a primary (usually glandular) focus of disease elsewhere. This is true, but I think that it is misleading to speak of a tuberculous hip or knee as a local manifestation of a general disease.

The normal pathological course of a tuberculous lesion tends to make it extravascular, and, except in cases of multiple and miliary tuberculosis, where the defensive mechanism has completely broken down, it would seem to be extremely improbable that " showers of tubercle bacilli " are poured into the blood-stream with any degree of frequency. Rather would I suggest that tuberculous arthritis is a metastatic infection due to the accidental detachment of a minute tuberculous embolus in the course of the extension

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of the primary lesion. Except in the cases of general dissemination that I have mentioned, such an accident is not likely to be repeated often, and the comparative rarity of multiple joint tuberculosis is in accord with this view. A tuberculous hip or knee is as much a local disease as is the primary focus, wherever that may be. It is true that the treatment of the one alone will not cure the other, but that does not mean that neither should be dealt with locally, if accessible.

We hear little of tuberculin nowadays, and it appears to be generally out of favour in this country ; but a few physicians to my knowledge are using it still in selected cases with good results. This brings me to the point that if tubercle bacilli are not regularly poured into the blood-stream, their toxins certainly are, and the effects of tuberculin thus self-administered are not unimportant.

A tuberculous focus of some sort, usually healed and encapsuled, is found post mortem in about 100 per cent of adult bodies. It is probable that the possession of such a focus is an advantage to most of us, because of the immunity which we develop from small doses of tuberculin. On the other hand, large excessive doses of tuberculin are known to be harmful and dangerous, since they lower the resistance of the body to infection and favour the spread of the disease. It is not unreasonable to suppose that the removal of one considerable area of tuberculous infection may have a beneficial effect upon another and perhaps less accessible focus of the same disease, as well as on the individual as a whole, by enormously diminishing the dosage of tuberculous toxin that is absorbed into the general circulation.

The pendulum swings. Twenty-five years ago we saw a reaction against the prevalent practice of so-called excision of the hip and its appallingly bad functional results. During the last twenty-five years we have seen the rise and fall of so-called conservative treatment, and its failure to cure tuberculous disease of the hip. To-day there is a reaction again in favour of more radical treatment in this disease. Some surgeons, notably Hibbs of New York, are doing arthrodesis of the hip in practically all cases, and some of our own surgeons are following the lead of Hibbs. It remains to be seen whether the abolition of movement at the hip-joint will cure tuberculous disease of the *pelvis*.

This brings me to one of the most important considerations in the pathology of tuberculous hip—namely, Pugh's observation that the disease begins commonly in the inner portion of the ilium immediately above the acetabulum, and that it spreads from that point to the non-articular portion of the acetabulum and through the ligamentum teres to the head of the femur. Tuberculous disease of the hip is, in fact, tuberculous disease of the pelvis, and if it does spread to the hip-joint, so that the affection of the joint dominates the clinical picture, that does not alter the fact that we are dealing with pelvic disease. The old so-called excisions of the hip were not excisions of the hip-joint at all, but consisted merely in removal of the head of the femur, leaving the principal seat of the disease untouched. Modern fusion operations, particularly those of the extra-articular or mainly extra-articular type, which do not entail disarticulation of the head of the femur, also do nothing to remove the principal seat of the disease in the acetabulum, and, theoretically at any

rate, by preventing the ascent of the femur, they must also prevent closure of the tuberculous cavity which always exists as the result of bone destruction.

I should like to compare these fusion operations on the hip with similar operations on the spine, for it seems to me that the principles involved are not dissimilar.

Tuberculous disease of the spine begins characteristically in the anterior part of the spinal column, and it leads to destruction of portions of two or more adjacent vertebral bodies. The cavity thus formed by the destruction of bone is filled with soft tuberculous material. Natural cure of spinal caries occurs, and can only occur, by collapse of the contiguous vertebræ. The vertebra or half vertebra immediately above the disease sinks down to the vertebra or half vertebra immediately below it, obliterating the cavity and squeezing out the soft tuberculous material, so that solid bone comes into contact with solid bone, and union can then occur between them. Anything which prevents collapse of the vertebræ in spinal caries will prevent obliteration and healing of the tuberculous cavity, and will therefore prolong the disease indefinitely. The older surgeons of a couple of generations ago knew this well, and they liked to see what they called a 'good deformity' in spinal caries, for they knew then that the disease would be soundly healed. Conversely, they disliked what they called 'flat backs' in spinal caries, for experience taught them that such cases ran a protracted course and were generally unsatisfactory. We learnt the same lesson again in the early days of spinal fusion, when Albee's operation was done with more enthusiasm than judgement in many cases before collapse of the vertebræ had taken place. I myself had experience of two such cases—both of them adults with disease limited to two lumbar vertebræ, and thought at that time to be ideal cases for spinal fusion. Both operations gave immediate satisfactory results, followed by recurrence of signs of active disease within a year, and, so far as I know, these cases have never healed. It is fortunately hard to prevent collapse of the vertebræ in spinal caries by any means short of operation, and the results of conservative treatment are therefore generally good in spite of some misguided efforts to prevent deformity.

The problem of tuberculous hip is not essentially different from that of spinal caries. The disease begins, at least in many cases, in the iliac portion of the pelvis immediately above the acetabulum, and by its extension to, and destruction of, the roof and floor of the acetabulum and the head of the femur, it leads to the formation of a cavity filled with soft tuberculous material, bounded above by the undestroyed bone of the ilium and below by what remains of the head or neck of the femur. As in spinal caries, the only way that such a cavity can heal is by collapse or bringing together of the adjacent bones so that the tuberculous material is squeezed out and solid bone comes into contact with solid bone. Here also anything which prevents collapse of the bones and obliteration of the cavity will lead to the establishment of a chronic tuberculous cavity—encapsuled it may be, and quiescent perhaps for years—but unhealed and a constant menace to the patient.

Thirty years ago Lorenz maintained that ankylosis with sound healing was the best result that could be obtained in tuberculous disease of the hip. He contrasted the poor results obtained by the usual methods of traction and

OPERATION.—I operated on Dec. 20, 1929, excising the acetabulum and all the adjacent infected bone. The ilium was divided horizontally at the level of the sciatic notch, and the rami of the pubes and ischium were divided below the disease. The femur was sawn across below the trochanters, and its upper end, which was a thin shell filled with caseous material, was removed. The upper end of the shaft was implanted on to the divided surface of the ilium (*Fig.* 324). No attempt was made to deal with the large chronic abscess cavity which extended into the inner side of the thigh. The operation wound was left widely open and packed from the bottom. Perhaps I may add that the incision was made through 3 in. of dense indurated tissue, and no normal anatomy was encountered until the bone was exposed. The limb was put up in plaster in abduction.

Now after two years this patient has a stable painless hip on which she can stand and walk without assistance. The hip can be flexed to 80° and there is also

FIGS. 325, 326.—*Case* 2. Skiagrams showing condition before and after operation.

considerable movement in other directions. There is still a little thin serous discharge from the two sinuses on the inner side and from the one where the wound was packed for many months. But I believe that these sinuses are in the indurated soft parts, and I do not believe that this patient has any active or latent tuberculous disease of the hip now. Her general condition might almost be described as robust. Incidentally, she declares that her leg is no shorter than it was before, and that she is quite comfortable in the same shoe that she wore before. This, of course, is due to the correction of the adduction deformity.

Case 2.—H. F., a woman aged 30. First attended the Royal National Orthopædic Hospital in 1916 at the age of 16, complaining of pain in the back after exertion. Her history was that she had tuberculous disease of the left hip at the age of 3, and she was treated for four years at the Sevenoaks Hospital for hip disease. Since then she has got about freely and has made no complaint of her hip.

It was noted that she was an old and apparently healed case of tuberculous hip, with 1½ in. shortening, and a well-marked dorso-lumbar compensatory scoliosis.

The pain in the back was of the aching character that is often associated with this type of scoliosis. There was no sign of active disease of the hip. She was ordered a spinal support, which relieved her pain, and she wore it for four years.

Ten years later (July, 1930) she again attended the Orthopædic Hospital, complaining this time of pain in the left hip. The hip was fixed in flexion and adduction. There was very little movement, and this was painful. X rays (*Fig.* 325) showed extensive disease of the acetabulum, extending into the ilium above it, and destruction of the head of the femur.

OPERATION.—I operated on Oct. 21. 1930. The acetabulum was excised with the adjacent part of the ilium, and the upper end of the femur was removed. The shaft of the femur was implanted on to the cut surface of the ilium (*Fig.* 326). The wound healed by primary union, and she was discharged on Feb. 9. 1931 (four months after operation), walking in a long plaster spica with the aid of crutches. A short spica was substituted for this in May. In June, 1931, this was removed, and

FIGS. 327, 328.—*Case* 3. Skiagrams showing condition before and after operation.

she walked without support of any kind. (*Note.*—I think that the period of fixation in this case was much too long; but, owing to the extensive removal of bone, I was afraid of a sudden displacement of the femur into the pelvis. Unfortunately the patient overheard a remark to this effect, and, as she admitted afterwards, it made her nervous and slow in trusting her weight on the limb.)

This patient now has a stable painless hip on which she can walk without assistance. There is movement up to 45° or more of flexion, and there is just under 3 in. of shortening.

Case 3.—F. de W.. a woman aged 23. First seen at her home on Sept. 3, 1931, where I found her in bed suffering from acute pain in the right hip, which had come on on the previous day. She was terrified of being touched, and a detailed examination was impossible. The hip was fixed by spasm in 30° flexion. There was no obvious swelling of the joint. The temperature was 99° She was given an injection of morphia. and was sent in an ambulance to the Middlesex Hospital. There she was given an anæsthetic and the limb was put up in plaster. X rays (*Fig.* 327)

showed extensive disease of the right hip-joint, with a large abscess cavity containing a sequestrum in the ilium above the acetabulum, and partial destruction of the head of the femur.

The patient denied any trouble with the hip in childhood. She first had pain in the hip two years previously after playing in a lacrosse match. She then had acute pain in the night, and was unable to walk in the morning. It gradually got better, but she has been lame ever since. In spite of this she still played games and was able to ride. During the last four months she wore a belt, but the pain gradually got worse, culminating in the last acute attack.

OPERATION.—I operated on Oct. 10, 1931. The ilium was divided transversely above the abscess cavity and the upper half of the acetabulum was removed, with part of the head and neck of the femur (*Fig.* 328). The wound was closed without drainage.

FIGS. 329, 330.—*Case* 4. Skiagrams showing condition before and after operation.

The limb was put up in a long plaster spica for four weeks, after which a short spica was put on, and the patient was discharged walking in this plaster on Nov. 18, 1931. The plaster was removed on Dec. 1 (note that this patient was walking in plaster five weeks after the operation, and without support of any kind in seven and a half weeks.) At the present time she has a stable painless hip on which she can walk without assistance. There is flexion movement to 40° or rather more, and about half the normal range of abduction, adduction, and rotation. She is comfortable with a 1½-in. lift under the heel. She uses a stick when she is out, but she walks indoors without one.

Case 4.—M. F., a woman aged 26. The patient was treated at the London Hospital for tuberculous disease of the left hip at the age of 8 years. She was in a Thomas splint for eighteen months. The disease was quiescent for two years and then she had a severe relapse. She was in a splint again for two years, and then walked on crutches for another year. She was then 15 years old. From the age of 18 onwards she worked in an office, and had no further trouble with her

hip until May, 1931, when, at the age of 26, she had sciatic pain and a large abscess presented in front of the hip. On examination the left lower limb was wasted and 4 in. short and there was a large abscess in front of the left hip. Flexion was free and painless up to 90°; other movements were practically absent. She had no pain except after walking, and then it was felt at the back of the hip and on the inner side over the adductor muscles. X-rays (*Fig.* 329) showed extensive disease with cavitation of the acetabulum and of the ilium above it. The disease also extended into the ischium. The head of the femur was destroyed. The patient was admitted to the Middlesex Hospital on Nov. 27, 1931.

OPERATION.—I operated on Dec. 5, 1931. The acetabulum and the adjacent parts of the ilium and ischium were excised, and part of the head and neck of the femur was removed (*Fig.* 330). The wound was closed without drainage, and the limb was put up in plaster. A short plaster spica was put on on Jan. 9, 1932, and

FIGS. 331, 332.—*Case* 5. Skiagrams showing condition before and after operation.

she was got up five weeks after operation. (This plaster was removed on Feb. 16, ten and a half weeks after operation.)

At the present time she has a stable painless hip on which she can walk without assistance. There is flexion movement to 90°, and full range of abduction, adduction, and rotation. There is 4 in. shortening (which she says is less than she had before).

Case 5.—H. G., a man aged 54, a tailor. The patient had tuberculous disease of the left hip with abscess at the age of 10 years. Sinuses discharged from the front and outer side of the thigh, and eventually healed, but broke out again from time to time. The last abscess previous to the present illness occurred twenty years ago at the age of 34.

On April 28, 1931, he attended the Middlesex Hospital complaining of recurrent attacks of pain in the left hip, cramps in the leg, and inability to walk. The hip appeared to be firmly fixed in 30° of flexion and adduction. There was 2 in. shortening. There were scars of old sinuses in the groin and on the outer side of the thigh.

No evidence of a recent abscess. He was ordered rest, and he disappeared into the country for six months.

On Nov. 10, 1931, he came back, complaining again of pain in the hip, and an abscess was found over the back of the great trochanter. X rays (*Fig.* 331) showed extensive disease of the acetabulum and head of the femur.

OPERATION.—I operated on Dec. 12, 1931. The acetabulum and the head of the femur were excised. A large abscess was opened, and a sequestrum was removed from the roof of the acetabulum (*Fig.* 332). The wound was closed without drainage. The limb was put up in plaster in abduction. On Jan. 22, 1932 (six weeks after operation), he was discharged walking in a short plaster spica. This was removed on Feb. 16. There was then about 10° of flexion movement at the hip. Subsequently a small sinus opened in the scar, which discharged slightly and the hip became practically ankylosed.

At the present time this patient has a stable painless hip on which he walks well without assistance. There is practically no movement. He is comfortable with a 1½-in. lift under the heel.
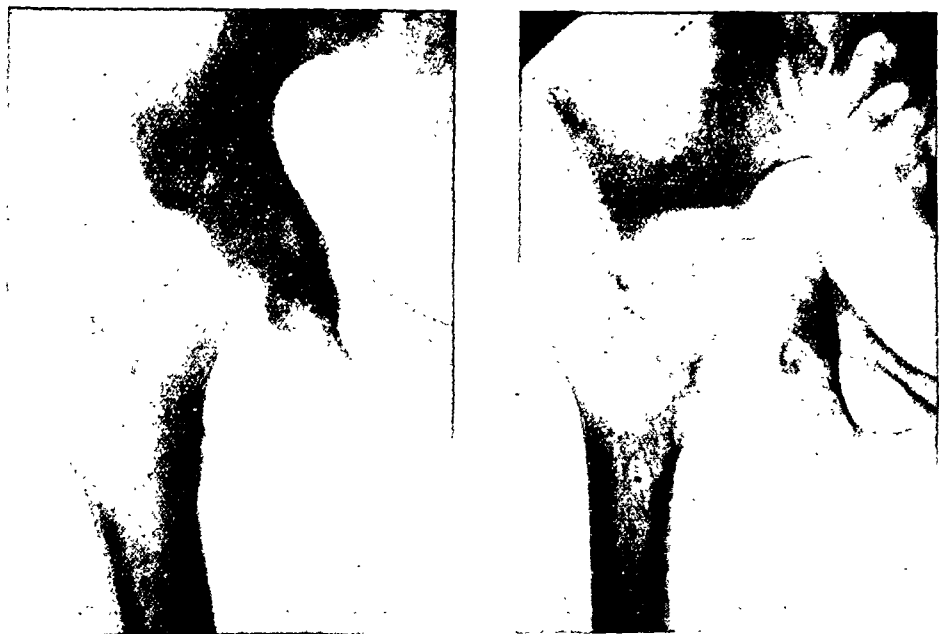
FIGS. 333, 334.—*Case* 6. Skiagrams showing condition before and after operation.

Case 6.—N. H., a woman aged 21. Attended the Middlesex Hospital on Dec. 9, 1930, complaining of pain in the right hip of about two years' duration. The pain had come on gradually when she was 18 or 19 years old, and it had gradually got worse. It was especially severe at night. There was no history of injury or of previous trouble with the hip.

On examination the right hip was held in a position of slight flexion and adduction. Flexion was possible from about 30° to 90°; all other movements were abolished. There was little wasting of the thigh muscles, marked wasting of the glutei, no obvious deformity or abscess about the hip, and half an inch or less of shortening. X rays (*Fig.* 333) showed destruction of the acetabulum with cavitation, and an indefinite outline of the head of the femur.

OPERATION.—I operated on Jan. 10, 1931. The acetabulum and part of the head and neck of the femur were excised (*Fig.* 334). A large abscess in the pelvis,

containing tuberculous pus and melon-seed bodies, was evacuated. The cartilage of the head of the femur was found detached and lying free in the joint. The wound was closed without drainage, and the limb was put up in a long plaster spica.

The patient was discharged walking in a short plaster spica on Feb. 28, 1931 (seven weeks after operation). This plaster was removed on June 16. At the present time she has a stable painless hip on which she can walk without assistance. There is movement up to 90° of flexion, and full range of abduction, adduction, and rotation. There is 2 in. of shortening.

Case 7.—B. W., a girl aged 16. Attended St. Bartholomew's Hospital in May, 1929, complaining of pain in the left hip. A diagnosis of tuberculous arthritis was made, and she was sent to the Sevenoaks Hospital for Hip Disease, where she was treated for three years. Eventually an extra-articular arthrodesis of the left hip

FIG. 335.—*Case 7.* Skiagram showing condition before operation.

was done at that hospital on April 28, 1931. During her convalescence after this operation she complained of stiffness and pain in the right hip, and she was admitted to the Royal National Orthopædic Hospital under my care on March 7, 1932.

On examination the left hip was firmly ankylosed in the fully extended position. On the right side all the movements of the hip were limited and painful, and there was muscular spasm and wasting of the thigh and gluteal muscles. X rays (*Fig. 335*) showed extra-articular arthrodesis of the left hip, and what we took to be early destructive arthritis of the right hip.

OPERATION.—I operated on March 15, 1932, excising the acetabulum and the head of the femur (*Fig. 336*). The wound was closed without drainage, and the limb was put up in a long plaster spica. The plaster was cut, and movements were begun three weeks after operation. The patient was got up and began to walk on May 10 (eight weeks after operation) without support of any kind.

At the present time this patient has a stable painless hip and she walks well in spite of the stiff hip on the other side. There is movement up to 45° of flexion on the operated side, and about half the normal range of abduction, adduction, and rotation. There is less than 1 in. of shortening.

Case 8.—S. G., a man aged 42. Complained of sudden onset of pain in the right hip in May, 1931. He had had no previous trouble with his hip. There was no history of injury. He was in a provincial hospital for several weeks with night sweats and a high temperature. His appendix was removed, but the pain continued. X rays (*Fig.* 337) showed rarefaction of the floor and inner part of the roof of the acetabulum.

After the patient's discharge the pain gradually increased in severity and he was re-admitted to the same hospital in January, 1932. He now complained of severe shooting pains down the right leg, and he was put in a Thomas's splint with extension. X rays (*Fig.* 338) showed an extension of the rarefied area in the floor and roof of the acetabulum.

He was transferred to the Orthopædic Hospital in March, 1932. On examination his general condition appeared good. There was no history of tubercle in his family. He denied venereal disease, and the Wassermann reaction was negative.

FIG. 336.—*Case* 7.   Skiagram showing condition after operation.

There was an indefinite swelling around the right hip, and the great trochanter was prominent. All movements of the joint were limited and painful, and he could not raise the limb from the bed. X rays (*Fig.* 339) showed subluxation of the head of the femur with erosion of its upper margin, and extensive disease of the acetabulum and adjacent part of the ilium.

OPERATION.—I operated on March 29, 1932. The capsule of the joint was nearly an inch thick, and the joint itself was filled with solid tuberculous material which was scooped out in lumps. The acetabulum and the head and neck of the femur were removed (*Fig.* 340). The wound was closed without drainage and the limb was put up in a long plaster spica. The front of the plaster was removed in two weeks, and movements were begun. He was got up without support of any kind on May 18 (seven weeks after operation).

At the present time this patient has a stable hip on which he can bear his full weight without pain, although he still uses two sticks for walking. There is movement to 45° of flexion, abduction and adduction about normal, and rotation to about half the normal range. There is a little more than 1 in. shortening.

Case 9.—H. S., a man aged 41. Had tuberculous disease of the left hip at the age of 6 years. He had several operations, and was in hospital until the age of 10. Since then he has had no trouble with the hip, except that it was stiff, for thirty years. In November, 1931, he complained of pain in the left knee. In January, 1932, he complained of pain in the left hip. On examination there was a well-marked flexion and adduction deformity of the hip ; practically no movement ; little pain in the hip ; considerable pain in the knee ; marked wasting of the thigh muscles ; scars of old operations about the hip. X rays (*Fig.* 341) showed unhealed old-standing disease of the left hip with destruction of the acetabulum and head and

FIGS. 337, 338.—*Case* 8. Skiagrams showing successive stages before operation.

FIG. 339.—*Case* S.
Later stage before operation.
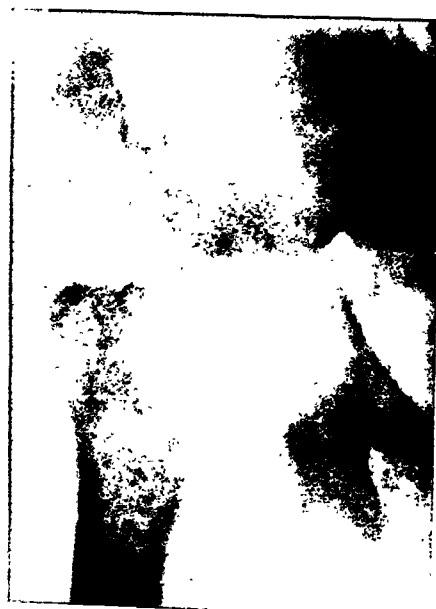
FIG. 340.—*Case* S.
Condition after operation.

neck of the femur. In February, 1932, a small abscess opened and left a sinus in one of the scars on the front of the hip.

OPERATION.—I operated at the Orthopædic Hospital on May 3, 1932. The acetabulum and the upper end of the femur were excised, and the upper end of the shaft of the femur was implanted on to the cut surface of the ilium (*Fig. 342*). The wound was closed without drainage, and the limb was put up in a long plaster spica. The plaster was removed in eleven days, and he was got up on crutches on June 16 (six weeks after operation). He was discharged on July 15 walking with sticks and with the boot raised 1 in.
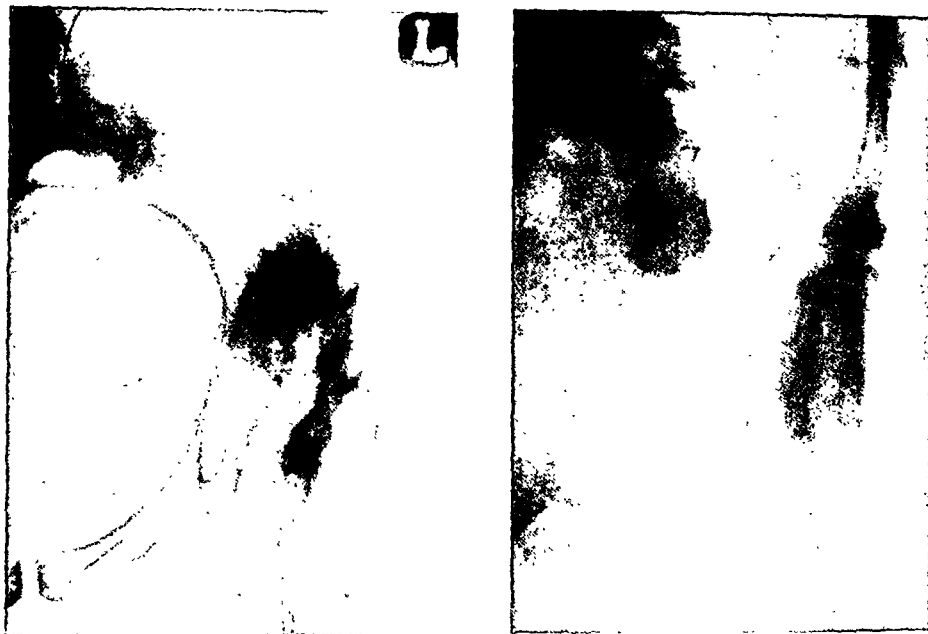
FIGS. 341, 342.—*Case 9. Skiagrams showing condition before and after operation.*

COMMENTARY.

It is not suggested that these recent cases represent final results, but I think they may serve as a basis for discussion and perhaps as a stimulus to further investigation.

It will be noted that the dominant feature of all these cases has been *pelvic disease*, quiescent or latent for years, but certainly never cured, and I would suggest that this is a common, if not the usual, result of the conservative treatment of tuberculous hip. In all these cases I believe (though I cannot prove) that the local disease has been removed completely.

With the exception of the first case with secondary infection, all healed by primary union. *Case 5* later developed a sinus, which discharged for a few weeks, and it is perhaps noteworthy that this is the only case in this series in which ankylosis occurred. With the exception of the first two cases also the period of convalescence has been remarkably short, the patients being got up and walking in from five to eight weeks after operation. All the patients

now have stable painless hips, and, with one exception, at least some degree of useful movement.

One cannot help wondering whether these patients would have been any the worse off, or whether they would not have been spared many years of hospitalization, chronic invalidism, and recurrent disability, if they had had their disease removed at the beginning, instead of at the end of their illness. In other words, now that the bubble of conservatism has been pricked, and the futility of striving for a literal cure has been exposed, might not a more rational surgery be applied to the treatment of tuberculous disease of the hip in children ?

To many, no doubt, the suggestion that we should return to radical surgery in the treatment of tuberculous hip will appear reactionary and horrible. We shall be told again that tuberculous hip is a local manifestation of a general disease, and that local surgery will not cure it. We shall be reminded that operative treatment has been extensively tried in the past and has proved a failure. From some quarters we shall hear again of the wonderful cures that result from open air, heliotherapy, and general conservative treatment, and the exceptional case will be put forward to prove it.

We have thought of all this, and we are still unconvinced. We do not believe that tuberculous hip is a local manifestation of a general disease. The inadequate operations of the past have nothing whatever to do with present-day surgery. The results of conservative treatment, as we know them, are profoundly disappointing, and I would submit that the time has come for a general reconsideration of the methods of attack in this disease. I would suggest that early excision of the focus of disease in the ilium might be considered as a rational method of treatment, and my experience of late cases leads me to believe that many cases might be cured at an earlier stage, and with a good prospect of preserving useful movement at the hip.

The operation is admittedly a severe one, requiring careful selection of cases and adequate precautions against shock. A reasonable rapidity in operating should be achieved, and I regard a blood transfusion running concurrently with the operation as essential. With these precautions the operation has as a rule been remarkably well borne.

Since this paper was first written I have had two disappointments which must be recorded. The first was a boy, 8 years old, with tuberculous disease of the left hip. At operation the disease turned out to be much more extensive than was anticipated, and at one stage there was troublesome bleeding from the internal pudic artery. The patient died a few hours after operation. The second case was a girl, aged 13 years, with tuberculous disease of the right hip. The operation was uncomplicated, but the patient died suddenly a few days later from pulmonary embolism—an accident which I do not know how to anticipate or prevent.

ACCIDENTAL LIGATION OF THE HEPATIC ARTERY.

REPORT OF ONE CASE, WITH A REVIEW OF THE CASES IN THE LITERATURE.*

By ROSCOE R. GRAHAM AND DOUGLAS CANNELL, TORONTO.

ACCIDENTAL ligation of the hepatic artery is fortunately rare. Twenty-seven cases have been reported in the literature up to date. The rarity of the lesion and the complete investigation carried out prompts the presentation of this report.

CASE REPORT.

W. W., male, age 49, was admitted to Dr. R. R. Graham's service of the Toronto General Hospital on Nov. 5, 1930, with a right inguinal hernia, hæmorrhoids, and a slight prolapse of the rectum. During the course of routine history and physical examination, a pyloric carcinoma was diagnosed and confirmed by radiological examination. Accordingly on Nov. 12, 1930, following the usual pre-operative preparation, a partial gastrectomy was done.

A moderately extensive pyloric carcinoma was found, with enlarged lymph-glands extending upward into the gastro-hepatic omentum and along the lesser curvature of the stomach, almost to the œsophagus ; glandular enlargement in the gastrocolic omentum was not extensive. There was marked inflammatory reaction and induration of the gastro-hepatic omentum which extended along the anterior border of the foramen of Winslow. The pylorus was freed after division of the gastrocolic omentum. The vessels along the superior border of the pylorus were isolated, divided, and ligated. During this procedure a large vessel which was enmeshed in inflammatory adhesions was divided and tied. It was immediately thought that the hepatic artery had been severed. Subsequently more careful examination following the closure of the duodenal stump confirmed this belief. Approximately one inch of the hepatic artery had been removed at a point about one and a quarter inches from its origin in the cœliac axis. The operation was completed in the usual manner, a posterior end-to-side retrocolic gastrojejunal anastomosis being done. No changes were noted in the liver during the operation. The abdomen was closed in layers without drainage, and the patient returned to the ward in good condition. Post-operatively continuous intravenous saline was given by the drop method, supplemented with the usual sedatives. Blood-sugar (*Fig.* 343) and non-protein nitrogen estimation (*Fig.* 344) were done hourly for the first four hours and at greater intervals thereafter. These showed no marked variation from the normal. The Van den Bergh reactions were negative throughout, and small quantities of urobilin were detected in the urine only the day preceding death.

Clinically the patient progressed favourably for the first three days. The evening of the fourth day his temperature rose to 103° F. with a simultaneous eleva-tion in pulse and respirations. Bilateral bronchopneumonia was diagnosed. He became rapidly worse and died on the seventh day of pneumonia, cardiac failure, and terminal pulmonary œdema.

Post-mortem examination revealed extensive bilateral bronchopneumonia of

both lower lobes with an associated œdema of the remaining lung substance. The pathological findings in the heart and other organs exclusive of the liver were not remarkable. A moderate degree of diffuse peritoneal exudate was present.
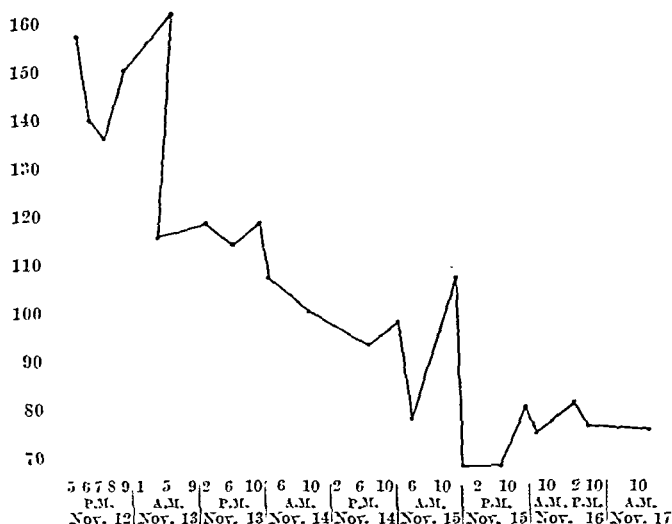
FIG. 343.—Blood-sugar estimation.
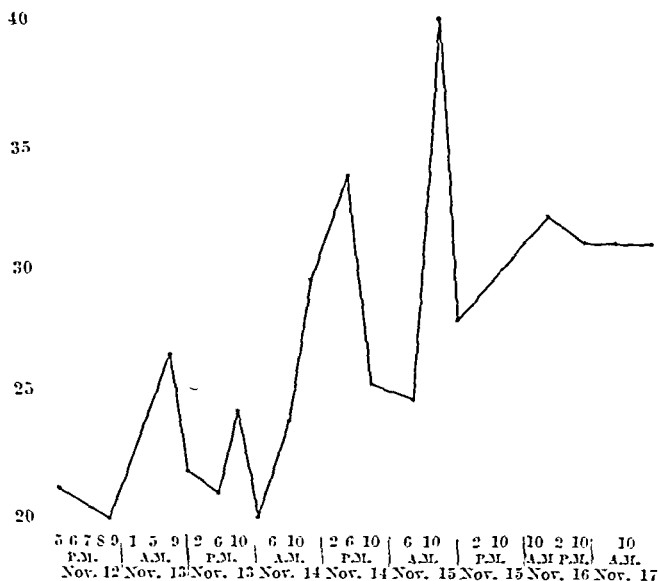
FIG. 344.—Non-protein nitrogen estimation.

The anterior and posterior aspects of both lobes of the liver were covered with several fibrinous plaques. The left lobe presented a depression on its anterodorsal surface which measured about 7·5 × 5·5 cm. This was paler in colour and softer in consistency than the surrounding liver substance. The whole left lobe appeared

softer than the right, which seemed healthy. Several irregularly-shaped pale areas were seen on the cut surface of the left lobe, the largest of which measured 4·2 by 1·7 cm. They were yellowish-white in colour ; the lobular markings were indistinct and well demarcated from the remaining liver substance by a slight band of hyper-
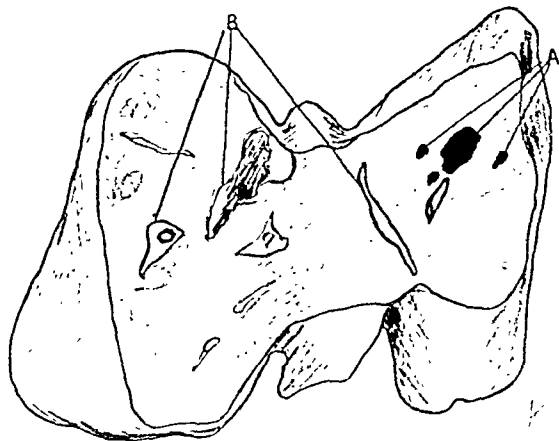
FIG. 345.—Sketch of cut surface of liver. A, Extent of necrosis in the liver ; B, Normal liver vessels.

FIG. 346.—Microscopic appearance of the necrotic area.

FIG. 347.—Showing the margin of the area of necrosis in the subcapsular region.

æmia. This necrosis did not involve the capsule or the liver substance immediately beneath it. (Fig. 345.)

Elsewhere in the lobe the liver was a dark reddish-brown colour and the lobular markings stood out well. Microscopically the appearance varied, depending upon the

area from which the section was obtained. Some showed normal liver cords with their usual well-defined arrangement. Others presented varied degrees of necrosis where only hazy outlines of hepatic architecture remained; the liver cells were jumbled together in irregular masses of pale-staining granular cytoplasm, nuclei being conspicuous by their absence (*Fig.* 346). Toward the periphery of these areas the liver cells were fragmented and pale-staining; the bile-ducts stood out prominently in comparison. Although all the liver cells undergoing necrosis were involved to a greater or lesser degree, those immediately beneath the capsule appeared slightly healthier than those in the more central zones. The capsule was thickened and an increased deposit of fibrinous material was present on its surface, in which a few small capillaries could be seen (*Fig.* 347). Minute, careful examination of the vessels showed that the only possible sources of arterial blood-supply were through the anastomoses of the phrenic arteries in the diaphragm, and possibly through a small anastomosis between the left gastric artery near the œsophagus and the left lobe of the liver. The latter seemed precarious, and it is doubtful if any arterial blood was carried to the liver from this source.

The relatively small amount of liver necrosis in this case was not sufficient to cause death. There were no clinical signs or symptoms of its existence, and no evidence to make one believe that death was due to liver insufficiency. The laboratory findings corresponded with the clinical course. The blood-sugar ranged from 0·15 to 0·08 mgrm. per 100 c.c. of blood, while the non-protein nitrogen varied from 22 to 39 mgrm. per 100 c.c. of blood. The Van den Bergh reaction was negative, and urinalysis showed a trace of albumin, moderate ketosis, and faintly positive urobilin reaction. We have reason to believe from the reports previously published that had he an otherwise uncomplicated post-operative course, our patient would have survived the damage done to his liver by ligation of the common hepatic artery.

REVIEW OF THE LITERATURE, AND DISCUSSION OF ARTERIAL BLOOD-SUPPLY TO THE LIVER.

Ligation of the hepatic artery has been reported by Socin,[1] Salzer,[2] Ritter,[3] Kehr,[4] Sprengel,[5] Tuffier,[6] Wendel,[7] Behrend,[8] Alessandri,[9] Bertram,[10] Tichow,[11] Kausch,[12] Narath,[13] Palacio-Ranam,[14] Wilms,[15] Gurbe and Herrenschmidt,[16] Von Haberer,[17] Klose,[18] Holst,[19] Bakes,[20] Smith,[21] and Hofmeister.[22] These have been divided into five groups by Ritter[3] in his excellent review of this subject—namely : (1) Ligation of arteria hepatica communis with ligation of collateral circulation ; (2) Ligation of arteria hepatica propria ; (3) Ligation of the right hepatic artery ; (4) Ligation of the left hepatic artery ; (5) Cases where point of ligation was not clear. In order that this classification may be better understood, the blood-supply to the liver is briefly reviewed as follows :—

The liver is supplied with blood from two great sources : the portal vein and the hepatic artery. The ramifications of the former and its part in the maintenance of liver viability and function do not enter into the scope of this report. Suffice it to say that there is a wealth of experimental evidence to prove that oxygenated arterial blood is essential to the maintenance of healthy liver tissue (Baruch,[23] Behrend,[8] Bainbridge and Leathes,[24] Segall,[25] Cameron and Mayes,[26] and Polettini[13]). The arterial supply to the liver is derived mainly from arteria hepatica communis, which arises from the cœliac

axis. As Segall points out in his study of the blood channels of the liver, the anastomoses of the phrenic arteries with the hepatic artery may be sufficient to carry on the circulation of the liver should the latter be occluded. We could find no direct branch of the inferior phrenic arteries which went to the liver, but the anastomoses through the diaphragm probably were sufficient. Extensive anastomoses are normally present which permit marked damage to portions of the arterial supply of stomach, liver, and spleen without impairment of their substance. From *Fig.* 348, which shows the normal collateral circulation in connection with the hepatic artery, it can be seen that the liver could be supplied satisfactorily through the left gastro-epiploic, the left gastric, and possibly through the phrenic arteries. In addition to the collateral circulation the presence of developmental anomalies which Behrend[27] found to exist in $33\frac{1}{3}$ per cent of his cases, and the fact first enunciated by Ruysch[28] that no given artery distributes itself in exactly the same manner in any two or more cases, make deliberate, accidental, and successful ligation
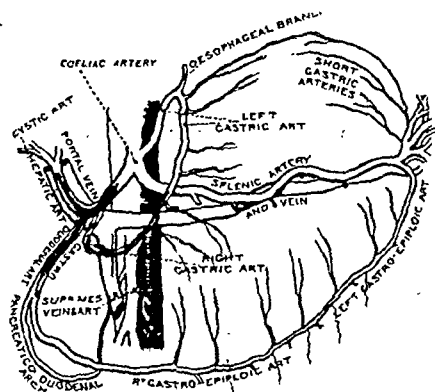
Fig. 348.—Normal arrangement of collateral circulation in connection with hepatic artery. The shaded area indicates damage in the present case. (*Cunningham.*)

of the vessel possible, as well as readily understandable. Three of the many possible anomalies are shown in *Figs.* 349–351. The course and distribution of the hepatic artery subsequent to its division into right and left hepatic arteries are well described by Segall.[25] He points out from his studies with experimental injection of human livers that the tying off of the right hepatic artery should have *no effect upon the liver substance so supplied if the left hepatic were intact, and vice versa*. He also notes that certain of the branches of these arteries are end arteries and their occlusion would result in infarcts. Such vessels terminate some distance beneath the capsule, and he accounts for the absence of necrosis in the capsule and immediately beneath it by the rich subscapular anastomoses.

Of the 27 cases previously reported, 15 died. A brief summary of these cases with their clinical and pathological findings will be found in the table on pp. 572–576. A large number of these have been abstracted directly from the papers of Ritter,[3] Rolland,[29] and Freedenwald and Tannenbaum.[44]

In addition to the cases reviewed, Kading[15] reported the successful ligature of the vessel by Sudeck to relieve an intra-hepatic aneurysm. Cases have been operated upon for the treatment of aneurysm of this vessel by Riedel,[30] Habs,[31] Friedman,[32] Merkel.[33] Baruch,[23] Anderson,[34] Sauerteig,[35] Niewerth,[36] and Miculicz.[46] In none of these, however, was the vessel
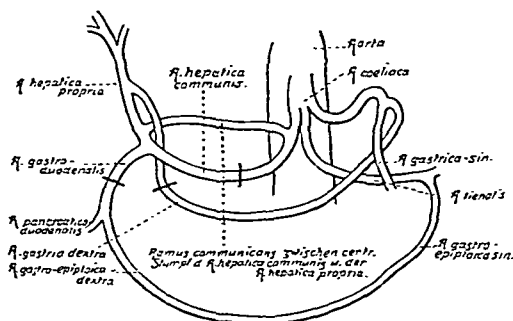
FIG. 349.—Anomalies of hepatic artery. An accessory common hepatic arising from arteria hepatica communis near its origin. (*Ritter.*)
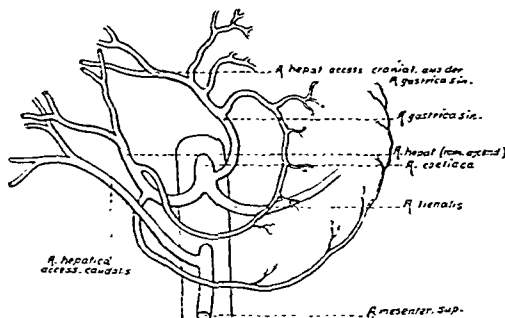
FIG. 350.—Anomalies of nepatic artery. Accessory hepatic artery arising from superior mesenteric artery. (*Ritter.*)
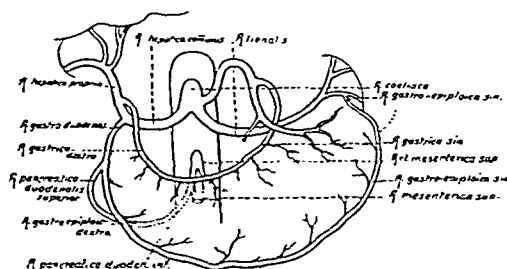
FIG. 351.—Anomalies of hepatic artery. Right gastric arising from arteria hepatic propria instead of the arteria hepatic communis. (*Ritter.*)

ligatured. Chiari,[37] Merkel,[33] and LeDieu[38] reported cases in which complete occlusion of the hepatic artery had occurred. Chiari by embolism, Merkel and LeDieu by thrombosis. There was frank liver necrosis in the first, a normal liver in the second. and cirrhosis of the liver in the third. The fact that there was no frank necrosis in the latter two cases is explained by their well-established collateral circulation.

ANALYSIS OF TWENTY-EIGHT CASES OF LIGATURE OF THE HEPATIC ARTERY

No.	Author	Vessel	Reason for Ligation	Clinical Course	Laboratory Findings	Pathological Findings	Result
1	Socin	Arteria hepatica communis	Hemorrhage from pancreatico-duodenal in stomach resection	Death in 3 hours	None	None	Death in 3 hours
2	Salzer	Arteria hepatica communis	Ligatured by mistake during gastric resection in pyloric cancer	Death in 7 hours	None	Flabby liver. No definite liver necrosis	Death in 7 hours
3	Ritter	Arteria hepatica communis	Ligatured by mistake during difficult gastric resection for penetrating ulcer. Accessory hepatic artery found	Tired and weak for some time. Pulse and temperature normal 4th day after operation. No jaundice. No evidence of liver insufficiency. Well 10 months later	Intensive investigation 7th to 21st day revealed very little disturbance of CHO or protein metabolism. VDB very slightly positive 8th day	None	Recovery
4	Kehr	Arteria hepatica propria	Ligatured purposely for haemorrhage in operating upon an aneurysm of right hepatic artery	Patient slightly jaundiced on 8th day		Next day liver bloodless; 1 cm. incision produced no bleeding. 10th day right border necrotic. A strip 2 cm. wide was ultimately detached from this border	Recovery
5	Sprengel	Arteria hepatica propria	Deliberate clamping of vessel in region of cœlic axis for hæmorrhage from a ruptured liver. This was released in 7 days and venous bleeding controlled by tamponade	Temperature subnormal on 10th day, deeply jaundiced, with extrusion of liver sequestra followed by pyrexia. Discharge from wound and death 20th day	None	Arteria hepatica propria torn between right gastric and gastroduodenal arteries. Two perforations of the portal vein. Multiple liver abscesses. Red infarcts	Death in 20 days
6	Kehr	Arteria hepatica propria	During an operation upon a case with stone in the common bile-duct, the stone was removed, the gall-bladder excised and the liver drained. Deliberate	Eighth day secondary haemorrhage. Contiguum and necrotic liver tissue removed; slow healing. Four months later abscess burst through wound. Slow convalescence	None	Necrosis of liver	Recovery

No.	Author	Artery	Cause		Clinical course		Findings	Result
			ligation to control profuse haemorrhage from the cystic artery. Ligated prior to bifurcation					
7	Tuffier	Arteria hepatica propria	Ligation for aneurysm	None	Death in coma in 4 days. Attributed to liver insufficiency	None	Ligature close to gastroduodenal and right gastric. Liver atrophied. No necrosis	Death in 4 days
8	Wendel	Arteria hepatica propria	Ligation during resection for gastric cancer involving hepatoduodenal ligament	None	Death in 36 hours	None	Haemorrhagic infarct and nearly total necrosis of liver	Death in 36 hours
9	Behrend	Arteria hepatica propria	Tied both ends of arteria hepatica propria for haemorrhage following severance of artery in an accident	None	Normal course for 10 days. Jaundiced on 11th day. Progressive emaciation; vomited occasionally and died on 14th day apparently of liver insufficiency	None	None	Death in 14 days
10	Hofmeister	Arteria hepatica propria	Tied accidentally during gastric resection	None	Out of bed on 2nd day. Discharged 17 days after operation. Normal post-operative course. No elevation of temperature beyond 38° C.	None	None	Recovery
11	Alessandri	Right hepatic artery	Accidental injury necessitating ligature	None	Severe circulatory damage made evident by functional disturbance and copious discharge for months	None	None	Recovery
12	Bertrand	Right hepatic artery	Accidental ligation during cholecystectomy for cholelithiasis followed by haemorrhage with application of clamp to vessel	None	Bleeding ceased at once. Right lobe looked anaemic. Liver looked normal on the eighth day. Clamp removed on fourth day. Course uneventful. Bile secretion ceased from wound in 7 days	None	Anaemic lobe appearing normal in four days	Recovery

ANALYSIS OF TWENTY-EIGHT CASES OF LIGATURE OF THE HEPATIC ARTERY—continued

No.	Author	Vessel	Cause for Ligation	Clinical Course	Laboratory Findings	Pathological Findings	Result
13	Tichau	Right hepatic artery	Not given	Death in 3 days. No sign or symptom described	None	Liver necrosis is extending to within 3 cm. of the fissure between the lobes	Death in 3 days
14	Wendel	Right hepatic at point beyond the cystic artery	Preparatory ligation prior to resection of the right lobe for removal of an adenoma	Healing without biliary fistula after resection of the lobe. Death 8 years later. No P.M. reports	None	None	Recovery
15	Kausch	Right hepatic at point beyond the cystic artery	No ligation. Thrombosis following too extensive cauterization during a cholecystectomy	Death resulted. Cause not given	None	Very slight liver necrosis	Death
16	Ritter	Right hepatic at point beyond the cystic artery	Ligated during a cholecystectomy in freeing the neck of the gall-bladder and ligating the cystic duct and artery. The right hepatic was here a branch of the cystic and came off unusually close to the liver.	Normal. Afebrile on 4th day. Pulse 80. Tired and weak for a long time. Home on 25th day. Appetite good. No gain in weight. Seen again 9 months later in good health	Liver function tests 7–21 days. Disturbed CHO and protein metabolism. Amino-acids elevated. Galactosuria present when given per os. Urine: albumin trace. sugar trace. Acetonuria 7th and 8th days. Urobilin slightly positive throughout. Van den Bergh negative. Benzaldehyde: trace Indican test always positive	No findings. Disturbed CHO metabolism and high amino-acid contents taken as indication of liver necrosis and functional disturbance	Recovery
17	Ritter	Right hepatic at point beyond the cystic artery	Ligatured by accident during a gastric resection for carcinoma in attempting to extirpate the lym-	Normal post-operative course until 6th day, except continuous weakness and mild bronchitis. Sud-	None	Purulent localized peritonitis in epigastrium. Jaundice. Necrosis of right lobe of liver with anom-	Death in 5 days

			phatic glands			aly of the hepatic artery and ligature of the right branch	
18	Hofmeister	Right hepatic artery	Tied deliberately in order to resect right lobe	denly temperature to with chill and weakness the next day. This continued with high fever, pain in R.U.Q., resistance and rigidity below right costal margin. No jaundice. Sudden shortness of breath with rapid pulse and death on 12th day. Deeply jaundiced just prior to death	None	None	Death in 5 weeks
19		Left hepatic artery	Injured during resection of stomach	Small gall-bladder fistula 5 weeks later with cachexia and no sign of liver damage	None	Extensive necrosis of left lobe and lobe of Spigeli, with localized epigastric peritonitis	Death in 7 days
20	Palacin-Ramon	Left hepatic artery	Ligated as a preparatory measure to resection of lobe for removal of gummata	Death in 7 days. Clinical course not described	None	The amount resected is not clearly stated	Recovery
21	Wilms	Left hepatic artery	Ligatured accidentally during gastric resection	Healing following resection by means of tamponade	None	Extensive necrosis of left lobe with perforation of the hepatic artery or one of its branches immediately beyond the bifurcation. It is suggested that bleeding was due to ulceration of the vessel following its ligation	Death in 10 days
22	Grube and Herrenschmidt	Left hepatic artery	Ligatured during extirpation of hydatid cyst in lobus quadratus	Sudden death 15 days post-operatively from secondary hæmorrhage	None	Total necrosis of left lobe	Death in 4 days
23	Von Haberer	Left hepatic artery	Ligatured in resecting the left lobe to remove hydatid cyst	Death in 4 days of liver insufficiency	None	None	Recovery

Uncomplicated healing

Analysis of Twenty-eight Cases of Ligature of the Hepatic Artery—continued

No.	Author	Vessel	Cause for Ligation	Clinical Course	Laboratory Findings	Pathological Findings	Result
24	Klose	Left hepatic artery	Ligatured in resecting the left lobe for primary carcinoma	Death in 4 days with high fever and rapid pulse—cause given as endocarditis	None	None	Death in 4 days
25	Holst	Left hepatic artery	Ligatured accidentally during a gastric resection for carcinoma of stomach. It was mistaken for the gastroduodenal artery as it ran into a mass of adhesions and was retracted in an abnormal manner	Post-operative course normal until the patient suddenly began to grow weak and died 8½ days after operation: never jaundiced. Death attributed to liver insufficiency	Normal amino-acids and ammonia in the urine	Left lobe small and soft with areas of necrosis in it. Right lobe's normal appearance attributed to a small vessel in the ligamentum teres	Death in 8½ days
26	Graham and Cannell	Arteria hepatica communis	Accidental ligation in resecting carcinoma of pylorus. A large mass about pylorus explained failure to identify vessel	Uncomplicated for 4 days. On evening of 4th day bilateral bronchopneumonia, and died 7th day of pneumonia, pulmonary œdema, and cardiac failure	Faint urobilin in urine. Normal Van den Bergh. Normal blood-sugar. Normal N.P.N. in blood	Depression on left lobe of antero-dorsal aspect 7.5 × 5.5 cm. Irregular areas of necrosis on cut surface. Necrosis did not involve capsule	Death in 7 days
27	Bakes	?	During cholecystectomy forced ligation of arteria hepatica propria or one of its branches owing to hæmorrhage	Uncomplicated post-operative course during 14 days' observation	None	None	Recovery
28	Smith	?	On removing a stone from the common duct there was an alarming hæmorrhage, to control which he ligated the hepatic artery	Jaundice for 10 days, followed by rapid disappearance. Parietal sepsis developed and ventral hernia resulted; 6 months later recurrence was treated medically	None	None	Recovery

From a study of these cases and our own, it is evident that ligature of the hepatic artery in any part of its course may be followed by serious results. It may, on the other hand, be looked upon in certain instances with less anxiety than has hitherto been the case. The results of sudden ligation of the vessel where no previous impairment in its circulation has been present must in most instances be followed by liver necrosis. The degree of necrosis and the ultimate recovery of the patient depends upon the point where the ligation has been done and the condition of the collateral circulation. This study tends to confirm the findings of Von Haberer,[17] Kohle,[39] and Narath[13] that :—

1. Ligation of arteria hepatica communis can be undertaken without fear of impairment of hepatic nutrition (Merkel,[33] LeDieu[38]).

2. Ligation of arteria hepatica propria before the right gastric is given off usually results in no ill effects (Chiara,[37] Sprengel[5]).

3. Ligation beyond this point is attended with total or severe necrosis where the artery has previously been healthy. The existence of previous arterial disease, anomalies, and the establishment of efficient collateral circulation make the latter ligation possible.

The findings of almost constant necrosis in ligations of the peripheral right or left hepatic arteries make one doubt the feasibility of applying the inference from Segall's[25] injection experiment to the living subject. However, in our own case we feel that the source of supply must have been through the phrenic arteries and their diaphragmatic anastomosis which he demonstrated so beautifully.

Likewise the clinical and laboratory findings in man tend to show marked variations from ligations and experiments carried out on animals by Behrend,[8] Behrend, Radasch and Kershner,[40] Collens, Shelling, and Byron,[41] Bainbridge and Leathes,[24] and Cameron and Mayes.[26] These are due in part to the relatively richer and more widespread blood-supply in the animals, and to the fact that experimental investigation on other animals can never be applied *in toto* to man.

It is interesting to note that we suspected no evidence of liver necrosis from our study of blood-sugar and non-protein nitrogen values. This may be accounted for by the relatively small necrosis which occurred, and by the shortness of the period over which we were able to make our observations. The latter view is in part confirmed by the findings in Ritter's[3] cases, where changes in carbohydrate and protein metabolism were found only after the condition had been present for some days. One may point out, however, that in the cases presented where laboratory studies are available, the patients did not exhibit any evidence of hypoglycæmia. In those where a fatal outcome ensued, no evidence is submitted which points to hypoglycæmia as a predominant factor in this event. Thus again the clinical findings in man do not correspond entirely with the experimental evidence obtained in animals by Mann and Magath[42] and Collens et al.,[41] where death in coma resulted in a short time from exhaustion of glycogen supplies in the body. Ritter[3] found, however, in his second case that disturbances of carbohydrate metabolism did occur, as well as a marked rise in the amino-acid content of urine. These findings are borne out experimentally by Mann and Magath[42]

in their investigations of liver function. Ritter[3] believed that any increase in amino-acids greater than four to five times the normal values, in conjunction with a serious clinical picture, was justification for re-operation and resection of the affected portions. That this procedure is justifiable, or would be successful in any instance, has yet to be proved. The absence of jaundice in many cases is surprising and interesting; for the most part its intensity corresponds with the extent of the liver necrosis. Death resulted in 16 of the 28 cases in which the hepatic artery or its branches have been ligated. The part played by the resulting liver necrosis in these deaths is not easily determined. For the most part they occurred in patients suffering from other diseases, and frequently accompanied extensive intra-abdominal operations. In at least 7 cases death was attributed to liver insufficiency. However, the extensive liver necroses which may occur without fatalities encourage one, should ligation of the artery become necessary or occur accidentally during an operation.

CONCLUSIONS.

1. A case of ligation of the hepatic artery is here reported, together with a review of 27 cases previously reported.

2. Ligation of the hepatic artery is always a serious, but not necessarily a fatal, accident.

3. The prospect of liver necrosis increases steadily as the point of ligature moves toward the periphery.

4. The extent of necrosis and the absence of clinical and laboratory evidence of its existence in our case is remarkable.

5. The rather delayed development of liver insufficiency in man contrasts sharply with the findings in experimental animals, and make successful operative interference a possibility.

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THE PATHS OF GALL-BLADDER INFECTION: AN EXPERIMENTAL STUDY.*

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INTRODUCTION.

MUCH work has been done in an attempt to prove the source of infection found in cholecystitis and gall-stones, though the exact relation of the infection to gall-stone formation is still in dispute. But though it is generally accepted that an intramural focus is the commonest starting-point of the actual gall-bladder inflammation there is by no means the same agreement as to the origin of the causative bacteria. Rosenow (1916) and Wilkie (1927) have done much to prove the frequency of intramural infection and are mainly responsible also for the hypothesis of 'elective localization'; they suggest a bacterial embolus of peripheral origin and lay stress on the specificity of certain streptococci. Many authorities would exclude the intestinal bacteria as possible primary causative agents and regard them merely as secondary invaders. Gordon-Taylor and Whitby (1930, 1932), in a series of routine examinations, as well as in a review of the literature, have clearly shown that intestinal bacteria are those most commonly found in human cholecystitis—a view which has been confirmed by Williams and McLachlan (1930) and which was universally believed prior to the work of Rosenow and Wilkie. It may also be emphasized that Williams and McLachlan ascribe an intestinal origin to the majority of strains of streptococci isolated by them from the biliary apparatus. By inference, therefore, one is justified in believing in the intestinal origin of the majority of embolic foci in the gall-bladder, and even if the intestinal bacteria are dismissed as mere secondary invaders one is still faced with the problem of detecting by what route these organisms have reached the gall-bladder.

Many writers assume that the liver is an efficient bacterial filter and that organisms arriving in the liver from the portal circulation are promptly trapped and destroyed or excreted in the bile. Our experiments, though primarily designed to determine the route of infection in gall-bladder disease, also provide some evidence on the filtration power of the liver. We have set out to contrast the fate of bacteria injected into the portal and systemic circulation respectively. We have been concerned also with the fate of other substances such as Indian ink and emulsions of malignant tumours when similarly injected. From these experiments conclusions are drawn as to the probable route of infection in experimental cholecystitis.

METHOD OF INVESTIGATION.

The general plan of the investigation was to make cultures of the blood, bile, gall-bladder wall, liver, and other organs of rabbits after injection of organisms by various routes. The cultures were afterwards made at intervals varying from a few minutes to several weeks. The standard dose injected was 0·5 c.c. of an overnight meat broth culture of *B. welchii*. The same strain was used throughout the whole series. *B. welchii* was chosen partly because its presence in human gall-bladder infections had already been studied (Whitby and Gould, Whitby and Gordon-Taylor) and partly because of the ease with which it may be recognized in culture. During the course of the investigation, however, another advantage of the *B. welchii* became apparent—namely, the relative resistance of the rabbit to the organism. As the result of this resistance most of the animals could be allowed to survive until the experiment was concluded without obvious ill effects developing, thus allowing investigation of the routes traversed by the organism without involving the complicating factor of gross inflammatory changes.

For the intra-portal injections, the abdomen was opened under ether anæsthesia through a middle-line incision and the injection made into one of the veins in the mesentery of the small intestine ; leakage was prevented by ligaturing the vessel on either side as the needle was being withdrawn. Systemic injections were made into one of the ear veins. The blood was taken for culture from the ear veins and in a few cases from the heart. The tissues were taken for culture immediately after the animals had been killed by chloroform inhalation. The abdominal wound was reopened with the cautery, and the bile aspirated with a very fine needle through a small cauterized spot on the gall-bladder wall. If the bile was blood-stained, which only rarely occurred, indicating either a puncture of the liver or a laceration of the gall-bladder wall, the specimen was rejected. Of the gall-bladder, a portion of the free surface was taken for culture, carefully avoiding all tissue adherent to the liver. All cultures were made into meat broth medium and were examined twenty-four and forty-eight hours afterwards.

Results of Intra-portal Inoculations.—These are shown in *Table I*. Each rabbit received 0·5 c.c. of an overnight meat broth culture of *B. welchii* into a mesenteric vein.

Gall-bladder Wall.—Organisms were grown from the gall-bladder wall in all the 17 cases cultured within thirty minutes of the intra-portal inoculation. At the end of twenty-four hours a few negative cultures began to appear, and by the end of a week slightly less than half were positive.

Bile.—Only 2 out of the 17 biles cultured within thirty minutes of the inoculation were positive. The incidence of biliary infections rose to a peak at forty-eight hours (9 cases out of 26).

Liver.—The liver, as might be expected from its anatomical position and its relation to the reticulo-endothelial system, was the most constantly infected organ of those examined. In all the 29 cases cultured within forty-eight hours the liver was positive, and by the end of a week the incidence of positive cultures had only dropped slightly (8 out of 9).

Blood.—All cultures of systemic blood made within the first thirty minutes

after intra-portal inoculation were positive. At twenty-four hours also most of the cultures were positive. But by forty-eight hours the number of positive cultures had fallen to 3 out of 15, and by the end of a week to only 1 out of 8.

Table I.—RESULTS OF INTRA-PORTAL INOCULATIONS.

NO. OF ANIMALS	TIME AFTER INOCULATION	POSITIVE CULTURES FROM				
		Gall-bladder	Bile	Liver	Blood	Kidney
8	Immediate ..	8/8	1/8	8/8	8/8	—
9	10–30 mins. ..	9/9	1/9	9/9	9/9	—
15	24 hours ..	13/15	3/15	—	4/5	—
26	48 hours ..	20/26	9/26	12/12	3/15	3/3
9	1 week	4/9	2/9	8/9	1/9	6/9
3	5 weeks ..	1/3	1/3	1/3	0/3	—
70		55/70 (78%)	17/70 (24%)	38/41 (92%)	25/49 (51%)	9/12 (75%)

Note.—Figures for positive cultures: Numerator = Positive results. Denominator = Cultures made.

SUMMARY.—The experiments show that immediately after intra-portal inoculation organisms can be cultured from the liver, gall-bladder, and systemic blood. The bile, however, does not become infected with any frequency until forty-eight hours later, and then only in a minority of cases. By the end of a week the organisms have disappeared from the blood and are beginning to disappear from the gall-bladder, but are almost invariably still present in the liver, where they may remain many weeks or even months (in one case the liver still contained the organism nine weeks after inoculation).

Results of Systemic Inoculations.—These are shown in *Table II*. Each rabbit received 0·5 c.c. of an overnight meat broth culture of *B. welchii* into the aural vein.

Table II.—RESULTS OF SYSTEMIC INOCULATIONS.

NO. OF ANIMALS	TIME AFTER INOCULATIONS	POSITIVE CULTURES FROM				
		Gall-bladder	Bile	Liver	Blood	Kidney
4	10–30 mins. ..	3/4	0/4	4/4	—	—
6	24 hours ..	4/6	1/6	4/6	6/6	3/6
12	48 hours ..	6/12	4/11	11/12	6/12	5/12
1	9 days	0/1	0/1	—	—	—
1	19 days ..	0/1	0/1	—	—	—
3	5 weeks ..	0/3	0/3	0/3	—	—
27		13/27 (48%)	5/26 (19%)	20/25 (80%)	12/18 (66%)	8/18 (44%)

Table II shows that the time distribution of positive cultures in the tissues and organs examined after systemic inoculation is approximately the same as after intra-portal inoculation. There is, however, a fall in the total

incidence of infections in the gall-bladder and kidney. Thus one concludes that infection by the portal route is at any rate as easy as by the systemic.

Results of Intra-portal Inoculation with Diluted Doses.—In the experiments so far described the number of bacilli injected has been large, the organism content being of the order of 500 million per cubic centimetre. A series of experiments was next performed in which progressive dilutions of the standard were injected intra-portally varying from 25 million bacteria to 25. The results of culture of the systemic blood, liver, and gall-bladder of these cases five minutes after inoculation are given in *Table III*.

Table III.—QUANTITATIVE INTRA-PORTAL INOCULATION. CULTURES FIVE
MINUTES AFTER INOCULATION.

NO. OF *B. welchii* INJECTED	CULTURES FROM		
	Blood	Liver	Gall-bladder
25 million	+	+	+
2¼ million	+	+	+
250,000 ..	+	+	0
25,000 ..	+	+	0
2500 ..	+	+	0
250 ..	+	+	0
25 ..	+	+	0

It can be seen that in all dilutions the organism could be recovered from the systemic blood five minutes after the intra-portal inoculation. Even for small numbers, therefore, the liver is not an efficient filter and does not prevent the entry of some organisms into the systemic circulation. It will also be seen that with injections of 250,000 organisms or less, though the organisms reached the general circulation, the gall-bladder was sterile. The healthy rabbit's gall-bladder thus shows no special tendency to take up the *B. welchii* from the circulating blood when the numbers are small.

An attempt was next made to repeat some of the earlier experiments with smaller doses of organisms, and 8 rabbits were injected with a dilution of the original broth culture containing 2500 organisms per 0·5 c.c. The rabbit is. however, as has already been noted. so resistant to the *B. welchii* that at the end of forty-eight hours only 2 of the livers were found to contain the organism and none of the other tissues and organs examined (blood, bile, kidney. gall-bladder) were positive. In these circumstances experiments with the smaller dilutions were not continued.

DISCUSSION.

We will discuss these results as well as certain other experimental evidence in relation to the results of other workers and in relation to the theoretically possible routes of gall-bladder infection. namely : (1) By blood-stream infection—systemic or portal in origin ; (2) By lymphatic infection—from the liver or some other organ : (3) By way of the biliary passages—downwards

from the liver or upwards from the duodenum; (4) By direct spread from an abdominal viscus.

1. **By Blood-stream Infection.**—This is generally believed to be the most likely route of infection of the gall-bladder, strong evidence in its favour being the frequency with which, in cases of cholelithiasis, the gall-bladder wall is infected while the gall-stones and fluid contents are sterile. In our experiments, organisms could invariably be detected in the systemic blood shortly after intra-portal inoculation even when the dose injected was small. With the larger doses, the gall-bladder was constantly infected in the early stages when the bile was sterile, so that, leaving aside for the moment the question of lymphatic infection which we will discuss later, spread of organisms to the systemic circulation and thence through the cystic artery would appear to be the easiest route for infection to reach the gall-bladder even after intra-portal inoculation. Because the cystic artery is a probable route of infection of the gall-bladder, the inference is frequently made that some systemic focus of sepsis such as the teeth or tonsils is necessarily the source of the organisms. This view is expressed by Nickel and Judd (1930) and by Ogilvie (1929) and is clearly formulated by Illingworth and Dick (1932), who in their recent text-book state : " Organisms may reach the gall-bladder directly from the systemic blood-stream from some distant infective focus. This avenue of infection is the one most widely accepted at the present time although it is backed by no strong clinical support. On this view, septic foci in the teeth, tonsil or other regions provide the source of infection and it is usually believed that the organisms principally responsible are streptococci." The present experiments, however, show that the liver, like the lung, is quite inefficient as a bacterial filter and we would stress that experimental gall-bladder lesions produced by systemic inoculations could quite as easily have been produced as the result of intra-portal inoculation. Experiments with systemic inoculation thus prove nothing in the way of excluding the bowel as the origin of a bacterial embolus.

The intestinal nature of the majority of organisms found in relation to gall-stones has already been noted, and the possibility of organisms entering the portal circulation from the bowel is generally admitted, though the factors involved appear to be complex (Arnold, 1929). If it is admitted that intestinal organisms may pass out from the bowel, they may then readily traverse the portal circulation and liver to the general circulation, and one has to consider why they should lodge in the gall-bladder. In the present experiments when large doses were injected the gall-bladder in the early stages almost regularly contained the organisms, which probably had formed capillary emboli. In our experiments with the smaller doses, which approximate more to the conditions likely to obtain in man, the gall-bladder was sterile. One explanation of lodgement in the gall-bladder is the assumption of a specific affinity for the gall-bladder on the part of some organisms, as has been claimed for certain streptococci by Rosenow and Wilkie, though this has been denied by others (McLachlan and Williams) and is now being investigated by ourselves. Or, as an alternative explanation, it may be suggested that the gall-bladder at the time that the organisms are circulating happens to be in a state of lowered resistance which allows of their lodging.

If the view is taken that gall-stones are primarily aseptic and metabolic in origin, then the stones themselves may be responsible for the lowering of the resistance of the gall-bladder which allows of bacterial lodgement. If on the other hand biliary calculi result from the presence of bacteria in the gall-bladder wall, then some factor such as stasis or congestion has to be postulated to explain their lodgement in the first instance.

2. **By Lymphatic Infection from the Liver.**—The importance of infection of the gall-bladder from the liver through the lymphatics has been strongly urged by Graham (1929). Anatomically the natural direction of lymph-flow is from the gall-bladder to the portal fissure. There is no recognized route of lymph-flow from the liver to the gall-bladder. Thus on theoretical grounds a spread of bacteria from the liver to the gall-bladder would necessitate a reversal of the normal direction of lymph-flow. Our experiments show that the liver is much the most frequently infected organ at all times after inoculation. If therefore there were free lymphatic communication between the liver and the gall-bladder, one would expect the gall-bladder to contain the organism much more frequently than some other organ, such as the kidney, which did not possess this intimate lymphatic relationship. But the gall-bladder was not more frequently infected than the kidney, and, in fact, at the end of a week after intra-portal inoculation when organisms are beginning to disappear from most of the organs examined except the liver, the kidney was more frequently infected than the gall-bladder. So that the experiments so far as they go do not provide support for the lymphatic route of infection.

In an attempt to obtain further evidence on this point a number of rabbits were injected intra-portally with organisms after the gall-bladder had been separated from the liver and any direct lymphatic communications presumably divided. This, however, proved to be an unsatisfactory type of experiment from the technical standpoint owing to the changes produced in the gall-bladder through the injury necessarily inflicted and owing to the inflammatory reaction and adhesions produced around. Two different lines of experiment were therefore followed in an endeavour to obtain collateral evidence on this question of a possible lymphatic route of infection from the liver to the gall-bladder. Firstly, it is well known that Indian ink injected interstitially finds its way to the regional lymphatic glands (Cappell, 1929). Several experiments were therefore performed in which Indian ink was made to reach the liver in various ways. either by injection into the systemic or portal circulations, or by direct inoculation of the liver in the neighbourhood of the gall-bladder. After several days the animals were killed and microscopical sections made to include the gall-bladder and liver. In no case was there evidence of spread from the liver to the gall-bladder, though in all cases many cells filled with ink could be seen close under the capsule of the liver. A further series of experiments was performed in which Indian ink was injected as before, but in which in addition the gall-bladder was injured in various ways so as to produce an inflammatory reaction. But in these, too, there was no sign of spread of the ink from the liver to the gall-bladder.

The other way in which the intimacy of the lymphatic relations of the liver and gall-bladder was investigated was by the injection of tumour

emulsions. The Brown-Pearce transplantable carcinomatous tumour of rabbits is well recognized as spreading extensively by the lymphatics as well as by direct spread and by the blood-stream. By the injection of dilute tumour emulsions into the portal system with the same technique as was used for the *B. welchii* cultures, deposits of carcinoma were produced in the liver in several rabbits. And although these deposits were often widely spread throughout the liver, including that part of the liver most closely related to the gall-bladder, in no case were any deposits of growth present in the gall-bladder as would be expected if there were a free and active lymphatic connection between the two organs. These experimental findings with tumour emulsions merely serve to confirm a common observation in the post-mortem room, that even in *advanced cases of secondary carcinomatous* deposits in the liver the gall-bladder is extremely rarely involved—an observation that apparently also applies to primary malignant disease of the liver (Tull, 1932).

In face of such formidable negative evidence, therefore, the positive evidence that the lymphatic route from the liver to the gall-bladder is an important avenue of infection in human cases of cholecystitis and gall-stones requires to be *strong*. Graham's evidence consists largely in the demonstration of collections of inflammatory cells in the liver particularly around the intrahepatic biliary ducts. This 'pericholangitis', he thinks, may indicate an infection of the periductal lymphatics. But in rabbits at any rate this 'pericholangitis' is almost the rule, and in human livers, too, Flint has shown that it can very frequently be found even when there is no obvious evidence of disease of the biliary passages. It would appear, therefore, that Graham's evidence is *not sufficiently strong* and that his interpretations are capable of reversal—namely, that the cholecystitis precedes the hepatitis, especially since the hepatitis is more marked the nearer one gets to the gall-bladder (Wilkie[2]). In the absence, therefore, of strong positive evidence. the lymphatic route of infection from the liver to the gall-bladder cannot at present be regarded as important.

3. By Way of the Biliary Passages.—

a. Downwards from the Liver.—Our own results show that the injection of relatively large doses of bacteria, whether into the systemic or portal circulations, leads to excretion in the bile in approximately 25 per cent of cases. With smaller doses the incidence would probably be less. The excretion does not begin immediately but is quite definite at the end of twenty-four hours and is maximal in about forty-eight hours. The same result was obtained by Meyer (1921) using *B. typhosus*. We consider that our results give a true estimate of the amount of biliary excretion, for in that we used a relatively avirulent organism—*B. welchii*—with which there were no associated gross inflammatory gall-bladder changes, we were able to avoid the complicating factor of inflammation. With a highly virulent organism the gall-bladder wall may become inflamed and organisms be discharged from the inflammatory focus into the bile. Our results thus confirm those of Sherrington (1893). From these results one can only deduce that one of the methods by which the liver rids itself of bacteria is by excretion in the bile. and it is rational to assume that the bigger the implanted dose the more

constant is biliary excretion. One cannot therefore exclude biliary excretion as a possible route of infection.

b. Ascending from the Duodenum.—In that there is a natural open channel from the duodenum to the gall-bladder it was at one time supposed that this route would be a common one. This would be especially so when achlorhydria permits a heavy duodenal bacterial flora. The chief argument against this route is the frequency with which the contents of the gall-bladder are sterile when the walls are infected. But actual experimental evidence is scanty, and Meyer, who considered in great detail the other possible routes of infection, dismisses ascending infection from the duodenum very briefly and merely states that organisms introduced into the duodenum of rabbits could not be recovered from the bile.

The term 'ascending infection' is used in three ways. First it is used to describe a progressive infection of a duct system by contiguity of inflammation. For example, in some cases of inflammation of the female genital tract there may be successively a vulvo-vaginitis, cervicitis, endometritis, salpingitis. and finally pelvic peritonitis. From the absence of symptoms of duct inflammation in an ordinary case of gall-stones, however, it is unlikely that a similar sequence of events is common in the biliary passages. Secondly, some cases of apparent spread by contiguity of inflammation are believed to be examples of spread by periductal lymphatics, e.g., tuberculosis of the male genital tract. What is often understood, however, when ascending infection of the biliary passages is referred to is an infection from the duodenum along the lumen of the common bile-duct against the main biliary stream. By analogy with the urinary tract. the principal factor which would predispose to this would be some form of obstruction which damages the normal valvular mechanism of the duct opening and also creates a stagnant column of fluid in the duct system. An attempt was therefore made to reproduce these conditions experimentally in rabbits by partially occluding the duodenum a short distance below the entrance of the common bile-duct and injecting into the lumen of the bowel above the obstruction a culture of *B. welchii.* This was done in 6 animals. which were killed forty-eight hours later, and cultures immediately made of the bile, gall-bladder. and liver. In all cases there was considerable dilatation of the stomach and duodenum above the obstruction. The results of this experiment are given in *Table IV*.

Table IV.—CULTURES FROM RABBITS INOCULATED WITH *B. welchii* INTO THE LUMEN OF THE DUODENUM.

RABBIT	NO. OF ORGANISMS INJECTED	LIVER	GALL-BLADDER	BILE
1	500 million..	+	+	+
2	.. .. ..	+	+	0
3	.. .. ..	+	+	0
4	250	+	0	0
5	.. .. ..	0	0	0
6	.. .. ..	0	0	0
	Totals ..	4	3	1

It will be seen that of the 6 cases in only 1 was the bile infected with *B. welchii*, whilst the gall-bladder wall was in 3 and the liver in 4. In these experiments, in view of the sterility of the bile, an ascending infection in the bile can be excluded. And while the possibility of infection through the periductal lymphatics cannot be dismissed, from the previous experiments it seems likely that the gall-bladder and liver were infected through the blood-stream. These experiments therefore support the view that ascending infection from the duodenum along the lumen of the bile-duct must be a rare occurrence.

4. **Direct Infection from Neighbouring Viscera.**—Cases have occasionally been reported in which infection of the gall-bladder has occurred from an inflamed organ in the neighbourhood. For example, Rutherford (1930) has reported a case of cholecystitis secondary to a highly placed inflamed appendix. But these cases are chiefly reported as curiosities, and there is no evidence that organisms can spread in a similar way from non-inflamed viscera, so that this route need not be considered further.

CONCLUSIONS AND SUMMARY.

As the result of our experiments we have concluded that the cystic artery is the easiest route by which organisms can reach the gall-bladder even after intra-portal inoculation. We have already noted that the relative frequency with which in cases of human gall-bladder disease the gall-bladder wall is infected while the contents are sterile is generally believed to point to the cystic artery as the route of infection. Our experiments show that it is not a necessary inference from this that some systemic focus such as the teeth or tonsil is the source of the gall-bladder infection, as is sometimes asserted, and that the intestine affords just as likely a source. If in addition the well-recognized frequency of intestinal organisms in biliary infections is taken into account, it must be concluded that the intestine plays the predominant rôle in these infections. There is, however, one difficulty in the way of the acceptance of the blood-borne route of infection in human gall-bladder disease, and that is to explain why organisms should lodge particularly in the gall-bladder. The gall-bladder is not an organ like the spleen actively engaged in the taking up of organisms from the blood-stream. One explanation is the elective localization theory of Rosenow, which postulates a selective affinity on the part of certain organisms, particularly some strains of strepto-cocci, for the gall-bladder. This theory, however, has been strongly criticized (Williams and McLachlan). Otherwise one is left with the explanation that the gall-bladder is in a lowered state of resistance when the organisms are circulating. In this connection it is interesting to note that Andrews and Hrdina (1931) found after ligature of the cystic or common bile-ducts in dogs that an infection with intestinal organisms usually followed, though they attributed it to a lymphatic infection.

The only other route we consider worthy of serious consideration is that of excretion from the liver in the bile. Infection of the bile was present in 25 per cent of our experiments, though there is a latent period of some forty-eight hours before it becomes at all frequent. Excretion in the bile is pre-sumably one of the ways by which the liver rids itself of organisms, though there is some difference of opinion on whether the undamaged liver can allow

organisms to pass. Sherrington (1893) was of the opinion that damage to the liver cells had first to take place, but in our experiments organisms of a very low degree of virulence to the rabbit were excreted in this way. The biliary route would explain on an anatomical basis the special frequency of intestinal organisms in gall-stones, and would also fit in with the fact that the organisms commonly found are relatively bile-resisting, e.g., *B. coli, B. welchii, B. typhosus, Streptococcus fæcalis.* So that arbitrarily to dismiss this route as a possible path of infection, as is sometimes done, is not justifiable. The great objection to it, however, as we have already noted, is the relative frequency of infected gall-bladder with sterile bile, though this is more so in chronic cases than acute and in the former it is conceivable that the biliary infection has died out.

We may therefore summarize our findings and conclusions as follows :—

1. Experimentally, the cystic artery is the easiest route by which organisms can reach the gall-bladder.

2. The liver is not an efficient bacterial filter. Bacterial emboli may therefore reach the gall-bladder as easily from a portal focus as from a systemic one.

3. There is no evidence that lymphatic spread from the liver to the gall-bladder is a probable route of infection.

4. The possibility of infection of the gall-bladder by bile descending from the liver cannot be dismissed. Ascending infection by the biliary lumen from the duodenum is, however, probably extremely rare.

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ACUTE INFECTIVE OSTEOMYELITIS.
A REVIEW OF 262 CASES.

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THE series of cases reported here consists of all the cases of acute infective osteomyelitis which have been admitted to the surgical wards of the General Infirmary at Leeds during the ten-year period 1921 to 1930 inclusive. During this decade the number of surgical beds available has been constant, viz., 203, and the series of cases reported covers the work of all the surgical staff. In all, 262 cases are recorded, and of these 191 recovered and 71 died, giving a mortality of 27·1 per cent.

ETIOLOGY.

Yearly Incidence.—Although acute infective osteomyelitis is regarded throughout the country as a disappearing disease, the yearly incidence in this series is fairly constant, and there has been no real diminution in the number of cases (*Table I*). The area from which cases are drawn comprises a large part of the West Riding of Yorkshire, and includes rural localities, colliery villages, and both suburban and slum portions of the city of Leeds.

Table I.—YEARLY INCIDENCE OF ACUTE OSTEOMYELITIS.

YEAR	MALES	FEMALES	TOTAL
1921	10	4	14
1922	10	4	14
1923	28	7	35
1924	15	10	25
1925	12	13	25
1926	20	6	26
1927	23	9	32
1928	32	11	43
1929	15	10	25
1930	14	9	23
Total	179	83	262

Sex.—This series shows more than twice as many cases in males as in females, a proportion which agrees with that reported by Platt[1] in his series of 41 cases at the Ancoats Hospital (1923–7). In Fraser's[2] series of 400 cases there was a proportion of six males to one female, while Ogilvie[3] found a proportion of four males to one female in 51 cases. Choyce[4] states that the disease is three times as frequent in boys as in girls, while in Starr's[5] experience the disease is slightly more common in boys than in girls.

Age.—It is seen in *Table II* that the disease has a fairly uniform incidence between the ages of 5 and 14, there being 174 cases during these years and 88 cases outside them. Fraser[2] gives the maximum incidence in the 8th to the 12th years, while Platt[1] records 15 cases under 10 years of age, 19 cases from the 10th to the 15th year, and 7 cases over 15 years of age. Ogilvie[3] gives the average age as 12·9 years, and states that in 68 per cent of his cases the age was between 8 and 15 years. Starr[5] states that the majority of cases occur between the ages of 2 and 10.

Table II.—AGE INCIDENCE OF ACUTE OSTEOMYELITIS IN 262 CASES.

AGE	NO. OF CASES	AGE	NO. OF CASES	AGE	NO. OF CASES
Under 1	6	11	12	0–4	45
1	9	12	25	5–9	85
2	9	13	15	10–14	89
3	11	14	17	15–19	35
4	10	15	9	20 & over	8
5	17	16	13		
6	10	17	5		
7	17	18	5		
8	14	19	3		
9	27	20	2		
10	17	Over 20	6		

Site.—The most common sites of acute osteomyelitis in these 262 cases are the upper end of the tibia and the lower end of the femur, there being 62 examples in the former and 66 in the latter (*see Table X*). Next in frequency are the lower end of the tibia and the upper end of the femur, followed by the fibula and the radius.

Platt[1] gives the following figures from his series of 41 cases : tibia 13, femur 7, fibula 4, humerus 4, radius 3, ilium 4, ulna 2, metatarsus 2, and mandible 1. Fraser's[2] series of 200 cases shows a large preponderance of cases involving the tibia, and the incidence for the different bones is as follows : tibia 87, femur 32, humerus 20, ulna 18, radius 10, fibula 6, pelvis 6, tarsus 10, vertebræ 4, carpus 1, skull 2, and scapula 4. Starr's[5] figures coincide fairly closely with our own. Ogilvie's[6] figures for 51 cases are as follows : tibia 24, femur 13, humerus 4, fibula 4, ilium 3, os calcis 2, pubis 1, and metatarsus 1.

The great frequency of the disease in the neighbourhood of the knee-joint is to be explained by the frequent occurrence of strains, twists, and minor injuries in this situation and by the good metaphysial blood-supply and the size of the epiphysis. The frequency of acute osteomyelitis of the humerus in our series is much less than that recorded by other writers.

Predisposing Causes.—A history of trauma has been found in a relatively high proportion of cases in which inquiry has been made, although in some cases it is recorded that no injury has occurred (*Table III*). The length of time between the trauma and the onset of symptoms is usually less than a

fortnight. In many cases the trauma has been a blow, while almost equally often a history of a twist or a strain has been recorded.

Table III.—INCIDENCE OF TRAUMA.

		Cases
Trauma a 'few days' before onset	.. ..	10
,, 0–7 days	., ., ...	35
., 8–14 days	.. ., ...	20
,, over 14 days	.. ., ...	13
No trauma ..	.. ..	25
No record ..	.. ..	159
		——
		262

Beekman[7] states that in 60 per cent of his cases a history of trauma was forthcoming, while Ogilvie[6] records a history of injury in 18 of his 51 cases.

It has been very common to find a history of a recent exanthem or to discover that the child has been fretful and unwell for some days before the onset of pain. This prodromal stage is stressed by Platt.[1] Frequently one finds that the patient has recently suffered from a septic finger, a boil, or impetigo, or that such lesions are actually present at the time of admission to hospital.

Causative Organism.—In this series the causative organism has been the *Sta. aureus* in about 90 per cent of those cases in which bacteriological investigation was carried out (*Table IV*); this figure is higher than that given by most writers. There are no cases of osteomyelitis due to the pneumococcus, which figured prominently in Fraser's[2] series.

Table IV.—BACTERIOLOGY OF ACUTE OSTEOMYELITIS.

		Cases.
Sta. aureus ..	.. ..	66
Sta. aureus and *Str. pyogenes*	.. .. ..	2
Str. pyogenes	.. ..	3
Coliform bacilli ..	.. ..	1
Short-chained diplococcus	.. .. ..	1
No record ..	.. ..	189
		——
		262

Fraser[2], in a series of 400 cases, gives the following figures : *Sta. aureus* 78 per cent, *Sta. albus* 2 per cent, Streptococcus 6 per cent, Pneumococcus 14 per cent, *B. coli* 0·25 per cent, and *B. typhosus* 0·75 per cent. Ogilvie's[6] figures are as follows : *Sta. aureus* 42 cases, *Sta. aureus* with *B. proteus* 1, *Sta. albus* 1, *B. typhosus* 1 (51 cases). In Beekman's[7] cases the infection was caused by the *Sta. aureus* in 75 per cent and by the streptococcus in 10 per cent. The organisms in Lloyd's[8] 39 cases were : *Sta. aureus* 26, Streptococcus 10, Pneumococcus 1 ; while in Platt's[1] series of 41 cases they were : *Sta. aureus* 33 cases, Streptococcus 4, and *Sta. albus* 1.

PATHOLOGY.

Of the 71 fatal cases, 51 were submitted to autopsy, and of these all except 6 showed pyæmic lesions. Such lesions occasionally involved the lungs only, but were usually widespread, suppurative pericarditis and abscesses

in the kidneys being particularly common (*Table V*). In addition there are recorded 12 cases in which recovery took place after the treatment of pyæmic lesions, and it is probable that many more patients had small pyæmic abscesses in the lungs and kidneys which subsided without giving rise to symptoms. It is, therefore, important to realize that the onset of pyæmia by no means invariably necessitates a fatal issue. Fagge[9] states that chest complications, as indicated by pleuritic rub, pericardial friction, or cardiac murmurs, are very serious, and that in every case in which any of these signs have appeared, the result has been fatal. We can record one case of recovery after drainage of an empyema following acute osteomyelitis, and one of us (L. N. P.) can record a case which recovered after the drainage of a pyopericardium; this complicated an acute osteomyelitis of the upper end of the femur in a boy of 10 years of age.

Table V.—RESULTS OF AUTOPSY IN 51 CASES OF ACUTE OSTEOMYELITIS.

	Cases
Total number of autopsies	51
Acute pericarditis	33
Acute pleurisy or empyema	10
Abscesses or infarctions in lungs	22
Abscesses in kidneys	18
Acute arthritis	9
Metastatic osteomyelitis	4
Subcutaneous and intermuscular abscesses ..	9
Abscesses in myocardium	7
Abscesses in thyroid gland	1
Abscesses in spleen	3
Abscesses in prostate	1
Abscesses in liver	2
Acute duodenal ulcer	1
No pyæmic lesion found	6

Ogilvie,[6] recording the incidence of pyæmic lesions in his series of 51 cases, gives 11 cases of joint involvement, 9 of metastatic bone abscess, 3 of suppurative pericarditis, 1 case of meningitis, and metastatic abscesses as follows—myocardium 6, lungs 3, pleura 2, kidney 2, and soft parts 3.

Table VI shows the incidence of pyæmic lesions in which recovery followed treatment by drainage.

Table VI.—PYÆMIC LESIONS IN 12 CASES OF ACUTE OSTEOMYELITIS WHICH RECOVERED.

SITE	NUMBER	DETAILS
Bone	8	2 fibula, 2 humerus, 1 each tibia, radius, inferior maxilla, cuboid
Joints	3	Hip, knee, sterno-clavicular
Empyema	1	—
Suppurative pericarditis ..	1	—
Subcutaneous abscesses ..	6	Chest wall, buttock, etc.

Of the 6 cases which at autopsy showed no pyæmic lesions, 2 were cases of acute osteomyelitis of the ilium and of the lower end of the femur respectively. The former, in which death occurred three weeks after operation, was found on autopsy to have an acute suppurative sacro-iliac arthritis ; the latter died over three months after the first operation, following amputation of the leg necessitated by the development of an acute suppurative arthritis of the knee-joint. The other 4 cases died of septicæmia on the first, second, third, and fourth days respectively after operation ; they were all cases of osteomyelitis of the lower end of the femur.

SYMPTOMATOLOGY.

The length of history, as judged by the duration of pain, before admission to hospital was usually under seven days (*Table VII*).

Though in a surprisingly large number of cases the temperature on admission to hospital was less then 100°, the pulse-rate was 120 or more per minute in the majority of cases (*Table VIII*).

Table VII.—Length of History Before Admission to Hospital.

					Cases
' Few days ' pain before admission		..	..		14
0–4 days	,,	,, ..	..	..	70
5–9 ,,	,,	,, ..	..	..	35
Over 9 days	,,	,,	..	..	41
No record	..	..	..	..	102
					262

Table VIII.—Temperature and Pulse-rate on Admission to Hospital.

TEMPERATURE ON ADMISSION			PULSE-RATE ON ADMISSION		
Under 100°	..	87	Under 100 per minute	..	38
100°–101°	..	52	100–120 ,, ,, ..	..	64
101°–102°	..	37	120–140 ,, ,, ..	..	88
102°–103°	..	34	Over 140 .. ,,	..	43
103°–104°	..	18	No record	..	29
Over 104°	..	5			
No record	..	29			
		262			262

In cases of acute osteomyelitis of the long bones, the symptoms and signs are usually so typical that no difficulty arises in diagnosis. Platt[1] stresses the importance of recognizing a prodromal stage of the disease during which a small focus in the metaphysis is smouldering. This stage is characterized by fixed pain at the end of a long bone, and by tenderness on pressure of the metaphysis. In this stage also there is often an intermittent limp.

Acute osteomyelitis of the upper end of the femur is not always easy to recognize. A local sign of importance is tenderness over the greater

trochanter and in Scarpa's triangle, while should the hip-joint be involved, the limb is held in a position of flexion, abduction, and eversion, and the slightest attempt at passive movement elicits excruciating pain.

There is, except in the most fulminating cases, a leucocytosis of polymorphic cell type, the count often reading 25,000 cells per cubic millimetre. It must be emphasized that at this stage there is no radiological evidence of bone disease. Radiological signs do not appear until about the twelfth day, when some haziness of outline indicates periosteal reaction.

We should like to draw attention to the frequency with which osteomyelitis of the small bones of the foot mimics cellulitis of the foot. There have been eight such cases in our series. The whole foot is usually red, swollen, and acutely tender. After rest and local heat have been applied for a time, the infection becomes localized. Incision at this stage evacuates a small amount of pus or only some serous fluid, the condition being regarded as a cellulitis and subsiding in a few days. The wound, however, fails to heal completely, and a sinus remains. An X-ray examination made some weeks later reveals the changes resulting from an old acute osteomyelitis, and sequestra are usually found. In every case, therefore, of apparent cellulitis of the foot, especially if there is no obvious causative abrasion or wound, it is necessary to consider the possibility of infection of one of the smaller bones. One such case (included in this series) is recorded below :—

C. H. B., male, age 9, was admitted to Leeds General Infirmary on Oct. 19, 1926, with a swollen, red, painful left foot ; a diagnosis of cellulitis of the foot was made and the inner side of the foot was incised. A considerable amount of pus was evacuated, and the patient left hospital on Nov. 2, 1926, with the wound still draining. The sinus persisted, and in December, 1926, a radiogram showed a large sequestrum in the shaft of the first left metatarsal bone. On Dec. 11, 1926, the sinus was excised and the sequestrum was removed ; the boy made an uneventful recovery.

TREATMENT AND RESULTS.

Every case in the series has been subjected to operation, and particulars of the operations performed and of the mortality are given in *Table IX*. *Table X* gives the mortality according to the bone involved, and *Table XI* the time of death.

Table IX.—Operations Performed and Mortality.

OPERATION	DEATHS	RECOVERIES	TOTAL
Primary gutter operation	52	124	176
Primary partial diaphysectomy	1	19	20
Primary periosteal incision only	11	35	46
Primary periosteal incision : diaphysectomy later ..	1	2	3
Primary periosteal incision : gutter operation later..	4	11	15
Winnett Orr's method	2	0	2
	71	191	262

Table X.—BONE INVOLVED, WITH MORTALITY.

BONE			DEATHS	RECOVERIES	TOTAL
Femur	Upper end	..	7	15	22
	Lower end	..	22	44	66
	Not stated	..	3	1	4
Tibia	Upper end	..	24	38	62
	Lower end	..	3	24	27
	Not stated	..	2	5	7
Fibula	Upper end	..	1	7	8
	Lower end	..	1	14	15
	Not stated	..	0	3	3
Humerus	Upper end	..	0	7	7
	Lower end	..	3	3	6
	Not stated	..	2	2	4
Radius	Upper end	..	0	1	1
	Lower end	..	1	12	13
Ulna	Upper end	..	0	1	1
	Lower end	..	0	2	2
Ilium	..	..	1	1	2
Os calcis	..	..	0	4	4
First metatarsal ..		..	0	2	2
Second cuneiform		..	0	1	1
First phalanx, hallux		..	0	1	1
Mandible	..	..	1	0	1
Vertebral column, lumbar			0	1	1
Clavicle	..	..	0	2	2
Total	..	..	71	191	262

Table XI.—SHOWING TIME OF DEATH AFTER OPERATION.

TIME OF DEATH		NUMBER
Under 24 hours	..	5
1 day ..	..	7
2 days ..	..	7
3 ,, ..	..	5
4 ,, ..	..	6
5 ,, ..	..	3
6 ,, ..	..	2
7 ,, ..	..	2
8 ,, ..	..	2
9 ,, ..	..	2
10–14 ,, ..	..	6
15–20 ,, ..	..	11
21–28 ,, ..	..	6
Over 28 ,, ..	..	7
Total ..	..	71

The following is a brief description of the various methods of treatment as carried out in our series of cases :—

1. **Incision and Drainage.**—The end of the affected bone is approached by the orthodox route, and the subperiosteal abscess is incised for its full length and the pus is allowed to escape. The wound is left open, and treated usually by irrigation through Carrel tubes. Various antiseptics have been used, such as Dakin's solution, flavine 1–1000, hypertonic saline, and, more recently, quinanil. Occasionally the wound has been packed with gauze soaked in one of these substances. We have not been able to show that any one of these antiseptic substances has given better results than the others, and the antiseptic that has been used most is Dakin's solution. This operation has been performed in 64 cases. In 46 cases no further operation was needed in the acute stage of the disease, but the removal of sequestra was usually necessary later ; in a few cases a small flake-like sequestrum about one inch in diameter has separated after some weeks, and has been removed. Although simple periosteal incision with drainage has been performed for many cases of low virulence—including some of the cases of osteomyelitis of the small bones of the foot—it has also been performed on several patients who at the time of admission to hospital were too ill to allow of a more radical operation being performed. In the majority of such cases it was necessary some days later either to open the medullary cavity or to remove the shaft of the bone. In this series there were 18 cases in which a secondary operation was performed, and 13 of them recovered.

2. **Primary Diaphysectomy.**— This operation, which has been done in 20 cases with only 1 death, has only been performed in selected cases. There have been 8 cases for which partial diaphysectomy of the fibula has been performed ; this is a bone in which the extensive stripping of the periosteum, which so often occurs. suggests diaphysectomy as a simple method of treatment. In the fibula there has been no case of failure of regeneration of the bone. Diaphysectomy has been done six times for osteomyelitis of the radius, and in every case there was considerable late deformity from either partial or total failure of regeneration of the bone. This operation has also been performed twice for acute osteomyelitis of the tibia (amputation was needed later), once for acute osteomyelitis of the first phalanx of the hallux, three times for acute osteomyelitis of the clavicle, and once for the ulna.

3. **Gutter Operation.**—In our series this operation has been the routine procedure for the more acute cases, particularly in the femur and the tibia. The metaphysis is exposed and the periosteum incised. The compact bone is then removed by chisel for about one-third of the diameter of the bone. We regard it as particularly important that the removal of bone should extend right up into the metaphysis. In the other direction the bone is opened until healthy normally bleeding marrow is seen. Where little or no pus is seen. this extensive opening of the bone is modified. The wound is then either packed with gauze soaked in antiseptic or Carrel-Dakin treatment is instituted. In all. 176 cases have been treated by this method as a primary operative procedure. and 52 have died.

Two cases. in addition. were treated in accordance with the principles laid down by Winnett Orr. After the usual gutter operation the wound was packed with vaseline gauze and the limb was immobilized in plaster-of-Paris ; both these cases died.

COMPLICATIONS.

Apart from septicæmia and pyæmia, the most important complication in its danger to life is infection of neighbouring joints. This is to be expected in such sites as the upper and lower ends of the femur, and the lower end of the humerus, where the metaphysis is partly intra-articular. We have found, however, that the greatest incidence of joint involvement occurs in cases of acute osteomyelitis of the upper end of the tibia (*Table XII*). All writers stress the importance of the attachment of the periosteum to the epiphysial cartilage in preventing the spread of infection to the joint. We should like to emphasize the importance of carrying the incision in the periosteum right up to the epiphysial line, so that there may be no undrained collection of pus at this place; such a procedure is of especial importance at the upper end of the tibia, since the periosteum over the metaphysis is covered by the insertions of gracilis, sartorius, and semitendinosus, and by the internal lateral ligament of the knee-joint.

When an acute arthritis is established, harm is done by delay in treatment. Aspiration should not be persisted in if no improvement occurs. Arthrotomy should be done early, and, above all, amputation must be considered early. Many lives are lost through delay in deciding to amputate. Fagge[9] stresses the importance and seriousness of joint involvement, and says that it is his impression that no case in his experience complicated by a frankly purulent arthritis has escaped amputation at Guy's Hospital.

Amputation has been performed in this series in 12 cases (*Table XII*), in most of which acute arthritis of the neighbouring joint has complicated the osteomyelitis; in a small number secondary hæmorrhage has necessitated amputation.

Table XII.—INCIDENCE OF ACUTE ARTHRITIS AND OF AMPUTATION.

ACUTE ARTHRITIS	DIED	RE-COVERED	TOTAL	CASES OF OSTEO-MYELITIS OF BONE NAMED
Acute arthritis of knee from acute osteomyelitis of tibia	7	11	18	62
,, ,, of knee from acute osteomyelitis of femur	3	6	9	66
,, ,, of hip from acute osteomyelitis of femur	3	6	9	22
,, ,, of ankle from acute osteomyelitis of tibia	1	3	4	27
,, ,, of ankle from acute osteomyelitis of fibula	—	2	2	15
,, ,, of knee—pyæmic from acute osteomyelitis of fibula	—	1	1	—
,, ,, of knee—pyæmic from acute osteomyelitis of upper end of femur	1	—	1	—
Incidence of acute arthritis ..	15	29	44	—
AMPUTATIONS				
Acute osteomyelitis of lower end of femur	2	1	3	
,, ,, ,, upper end of tibia	2	6	8	
,, ,, ,, lower end of tibia	—	1	1	
	4	8	12	

DISCUSSION.

The mortality in this and in other recent series of cases is shown below :—

	Cases	Deaths	Percentage
Pyrah, L. N., and Pain, A. B., 1932	262	71	27·1
Gwynne Williams, 1932	91	18	19·8
Lloyd, E. I., 1932	40	13	32·5
Ogilvie, W. H., 1928	51	11	21·6
Mitchell, A., 1928	70	10	14·3

The recent work of Starr, of Toronto, has thrown new light upon the pathology of acute osteomyelitis. He has shown that infection spreads from the metaphysis to the subperiosteal space very early in the course of the disease, but that it spreads much later to the cancellous tissue of the shaft, and when such infection does arise it occurs not by direct continuity from the metaphysis, but by the entry of infection into the cancellous tissue from the subperiosteal abscess at different levels by way of the Haversian and Volkmann's canals. Thus, in the early stages of the disease the presence of a large subperiosteal abscess does not imply extensive medullary infection. Starr advised simple periosteal incision for those cases in which pus is found under the periosteum ; if no pus is found there, the metaphysis is to be drilled or trephined obliquely towards the epiphysial line.

It is a corollary of Starr's work that localization of infection will be most advanced at the metaphysis ; that in the diaphysis remote from the epiphysis there will be islands of infected cancellous tissue in gradually diminishing states of localization. It is a sound surgical principle that localized pus must be evacuated by incision and that the abscess cavity must be drained. The treatment of spreading unlocalized inflammation cannot be so dogmatically stated. Incision into an area in which such unlocalized, perhaps rapidly spreading, inflammation is present will on the one hand give exit to toxic material and to accumulating inflammatory débris, but on the other hand it must inevitably open up tissues which can allow of the rapid absorption of toxic material by veins and by lymphatics into the general circulation. In the treatment of acute lymphangitis it is now amply established by experience —particularly since the appearance of Kanavel's book[10]—that conservative treatment must be adopted until the lymphangitis has been controlled by heat and rest and that incisions into the primary focus of infection must only be made when pus has formed. The indiscriminate incision of such foci during the stage of progressive inflammation usually causes elevation of temperature and definite increase in the toxæmia of the patient. The treatment of cellulitis of the subcutaneous tissues is in general guided by similar principles. The day of multiple incisions for rapidly-spreading cellulitis has passed ; by rest and by the application of heat such localization of the infection can usually be obtained that one or two incisions made when pus has formed are all that is necessary. Such incisions, made when shutting-off of the infected zone has occurred, do not at all risk an increase of toxæmia, but promote its immediate and steady decrease. If the principles outlined here are applied to the treatment of acute osteomyelitis it would appear that those cases of great severity—whether from the great virulence of the infecting organism

or from the lack of resistance in the patient—should be treated by conservative methods such as simple periosteal incision; the bone should only be opened in those cases in which obvious and definite pus has accumulated.

We will now consider the series of cases here recorded in the light of the arguments advanced above.

In this series 64 cases have been treated by primary incision of the periosteum, and of these 16 have died (25 per cent mortality). Some of these have needed a secondary operation later. While many of these cases were mild cases in which the surgeon judged that the opening of the bone was not necessary and from whom a small sequestrum separated at a later date, very many were (to our definite knowledge) advanced cases in whom simple drainage was effected because the child was too ill to withstand radical drainage of the bone. Nevertheless the average results of this form of treatment are much better than those of the gutter operation. It is to be noted that the amount of sequestrum formation is much less than the area of compact bone from which the periosteum has been stripped.

The gutter operation has been performed (in this series) in most cases of average severity; there were 176 such cases in all, with 52 deaths. The mortality (29·5 per cent) of this group is considerably higher than that of the cases treated by primary simple periosteal incision. In performing the gutter operation the surgeon has usually removed compact bone from the region of the metaphysis along the shaft to a point where apparently healthy bleeding marrow has been observed, and definitely past the zone where oily (but usually not frankly purulent) marrow has been present. Inevitably therefore a considerable portion of marrow has been exposed where infection by way of the Haversian canals has but just occurred and where imperfect localization has been present; in doing this some blood-sinusoids have been opened which could allow general systemic absorption. We think that this is responsible for the higher mortality in this group of cases. The operation, undertaken to promote drainage of toxic exudation, must often have promoted exactly the reverse, namely absorption. Starr advocated the opening of the metaphysis by a small trephine or by a drill; this simple relief of tension in a rigid structure such as bone can scarcely be harmful provided that nothing further is done *if no pus is found*.

We think that the results of this extensive series of cases and the arguments we have put forward give support to what is definitely a strong tendency among most surgeons who have recently written upon acute osteomyelitis—namely, the more conservative treatment of the disease.

In a case of average severity the subperiosteal abscess should be incised for approximately its full length, and particular care should be taken to see that the incision reaches the epiphysial cartilage, since, as Starr showed, it is in the region of the epiphysial line that pus escapes from the cancellous tissue by one of the vascular foramina. The region of the metaphysis should then be explored either by a small trephine or by a drill. *If frank pus is found* upon such an exploration of the bone, compact bone should be removed only so far as to uncover the zone occupied by the pus and no farther. Provision should be made for drainage and for irrigation. From the few cases which we have seen treated by the method of Winnett Orr we cannot

advocate it ; too few cases, however, have come under our observation for us to be dogmatic about this method. *If no frank pus is found* upon exploration of the bone by trephine or drill, no further opening-up of the bone should be done.

If the patient is very ill or if the case is one of a very acute type in which a subperiosteal abscess of considerable size has formed within two or three days and in which the toxæmia is very profound, periosteal incision only should be done ; in some cases that survive it will probably be necessary to open the bone some days later.

SUMMARY.

1. A series of 262 consecutive cases of acute infective osteomyelitis is reported. Of these, 71 died, giving a mortality of 27·1 per cent.

2. Consideration of general principles, of the pathology of the disease, and of the methods of treatment employed in the cases in this series seems to point to the value of moderately conservative treatment for acute osteomyelitis.

We wish to thank the members of the Honorary Surgical Staff of the Leeds General Infirmary for permission to record the cases which have been under their care.

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'HYPERTROPHY OF THE PYLORUS' IN AN ADULT.

By P. T. CRYMBLE AND THOMAS WALMSLEY, BELFAST.

THE following case of 'hypertrophy of the pylorus' in an adult is worthy of record on account of the rarity of the condition, but even more so, we believe, on account of the remarkable arrangement of the parts which was discovered at the anatomical examination of them after their removal by operation.

The condition occurred in an adult male, age 43 years, married, and by occupation a labourer. He appeared to be, on general inspection, an ordinarily developed man though a little spare, and certainly he had none of the appearances of serious nutritional disturbance. He had had, he said, no gastric symptoms until he was 40 years of age, but more detailed inquiry from his relatives established " that when he was a small boy he was unable to eat fried eggs and meat and had to have light food, that he occasionally vomited, and that he was often troubled with indigestion", and also " that from the age of 16 years he was always careful in what he ate, as meat and potatoes did not agree with him." He had had, however, no medical attention for these complaints, and his other illnesses were unconnected with his present condition. Three years before admission, at the age of 40, he began to have gastric pain commencing about an hour after food, " burning sensations in the stomach, and waterbrash "; the pain was relieved by vomiting. The symptoms progressively became worse in spite of greater care in diet and occasional medical treatment, and there was some loss of weight ; at no time had blood been vomited.

FIG. 352.—' Immediate opaque meal ' : photographed in the erect position.

The opaque-meal examination showed " extreme 7-hour gastric retention " ; and obviously in the 'immediate' photograph (*Fig.* 352) there is obstruction at the pylorus and no filling of the first part of the duodenum. The stomach is of the J-shaped type, and the lowest point of the lesser curvature is below the intercristal line ; it appears, however, to have good

tone, even in the fundal part of the greater curvature, and though it is enlarged there is no more than a relative increase in the transverse diameter. It is considered now that the pyloric canal is empty, the possibility of a sagittally-placed first part of the duodenum having been taken into account.

At operation the stomach was found to be enlarged and its wall to be thickened. There was a firm 'tumour' at the pylorus of the size of a golf ball. It was covered with normal peritoneum, was freely movable, and to external examination seemed completely to obliterate the pyloric lumen ; it was recognized of course that its size and density precluded any estimate of the dimensions of the lumen. The commencement of the duodenum appeared to be normal in all respects. The lower half of the stomach and a small part of the duodenum were removed, and the alimentary canal was restored by a retrocolic end-to-side gastro-enterostomy.

The removed specimen consisted of a portion of the stomach 5 in. long, and included the lower part of the body and the whole of the pyloric part of the stomach and a narrow strip of the beginning of the duodenum. It had been slit along the lesser curvature. It was pinned out flat in Zenker-formol solution. The mucous membrane of the whole area, except over the pyloric mass, was slightly thickened, œdematous, and congested, and was covered with a small amount of mucus, but there were no signs of gross inflammatory or even catarrhal changes. In the area of the pyloric antrum there were four clotted surface hæmorrhages of bright red blood, two on the anterior and two on the posterior wall, and all of them much nearer the lesser than the greater curvature—that is, they were within the area of the *Magenstrasse*. Each hæmorrhage was circular in surface outline and about 1 cm. in diameter, and from each 'a stalk of blood' was continued into the mucous membrane through its surface. These hæmorrhages represent extra-vasations from the subepithelial plexus of mucous membrane veins.*

The examination and measurements of the cut surface of a block taken from the pyloric antrum demonstrate the conditions of general muscle hyper-trophy (the circular muscle is on an average 3 mm. thick and the longitudinal muscle 1·25 mm. thick), œdema of the submucosa, and slight vascular engorge-ment and œdema of the mucous membrane—that is, there is present a powerfully acting muscle wall of the stomach. Microscopic examination of this region demonstrates dilated capillaries in localized areas, and near the superficial surface of the mucous membrane, in the zone of the superficial venous network, there were numerous capillary hæmorrhages.

A longitudinal section through the pyloric end of the stomach, parallel with and close to the greater curvature, and a diagram which gives the

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* These veins are fairly large vessels. They are drained by rather fine vertical vessels which have little anastomoses and are valveless, and which connect the superficial and deep mucous membrane plexuses. The rupture of the superficial vessels in this region near the lesser curvature will be favoured by the much more close adherence of the mucous mem-brane to the muscle coat here than elsewhere in the stomach wall. The discussion of the mechanism of these hæmorrhages does not arise in this paper. It is of course well known that they occur in many abnormal gastric conditions, and their relation to ulcer formation has been freely discussed (see, for example, Disse, *Arch. f. mikros. Anat.*, 1904, lxiii, 512). Vomiting of bright-red blood in conditions of pure pyloric obstruction has been recorded by Bastianelli (*Ann. of Surg.*, 1925, lxxxi, 45).

dimensions of the parts, are shown in *Fig.* 353. It shows the great hyper-trophy of the circular muscle which formed the 'tumour', the sudden com-mencement of the hypertrophy, and that it exists in the whole length of and is confined to the pyloric canal. It shows, too, that the longitudinal muscle, which is 1·25 mm. thick in the antrum, is very much thinner over the 'tumour' —that is, is not hypertrophied, but rather in fact is less than normal in thickness. It also shows that in the pyloric antrum the mucous membrane is folded and the submucous layer is increased in amount and loose and
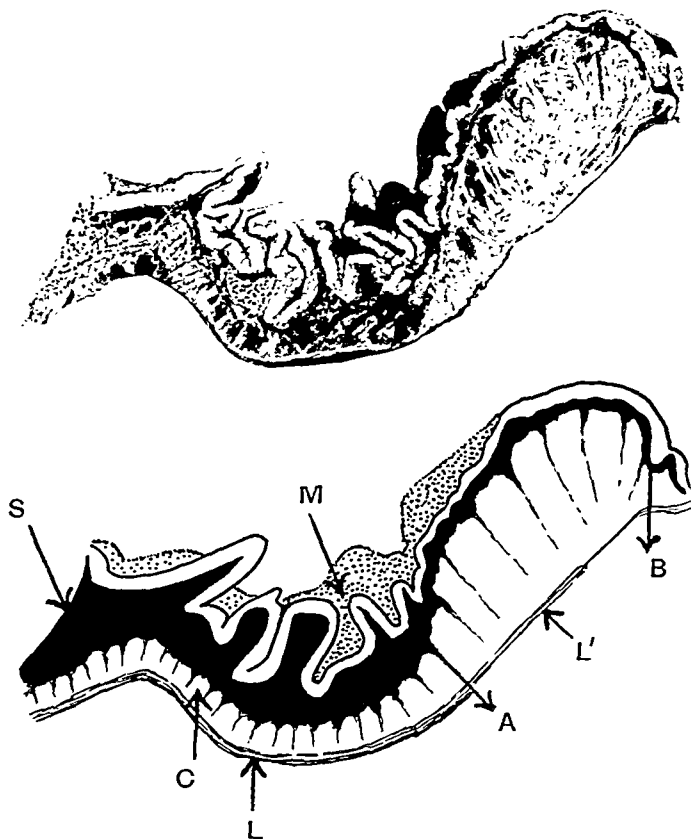
FIG. 353.—Photograph of a longitudinal section through the pyloric end of the stomach near the greater curvature. (*Natural size.*)
　　Below is a diagram to show the dimensions of the parts, after fixation. A–B=3·4 cm. (the length of the hypertrophied part at the lesser curvature was 2·5 cm.); C, Circular muscle, 3mm.; L, Longitudinal muscle, 1·25 mm.; L', 0·5 mm.; M, Mucous membrane; S, Submucosa. Greatest thickness of pyloric mass, 1·5 cm.

œdematous ; and that in this region there is a uniform and thickened circular muscle coat and a distinct and thickened longitudinal muscle coat. The mucous membrane over the 'tumour' is practically unfolded, and it is obvious that it is much more closely and firmly bound to the muscle ; and, as is shown in *Fig.* 354, the duodenal mucous membrane covers the whole of the duodenal face of the 'tumour'.

The hypertrophied mass consists of bundles of normal visceral muscle which are looser in their arrangement and embedded in a greater amount of fibrous tissue than is usual in smooth muscle (*Fig. 354*). The fibrous tissue is continuous with that of the submucous layer. In the internal parts of the mass the muscle bundles are almost entirely circular in direction, but more superficially they are oblique or vertical and here they freely interlace with one another; and these bundles are continuous with, and appear to be extensions of, a more or less normal layer of circular muscle which underlies the thin layer of longitudinal fibres on the surface. In the position where
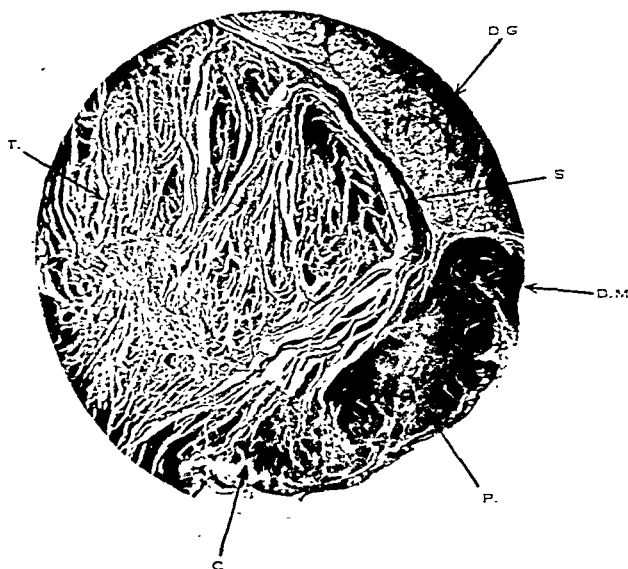
FIG. 354.—Microphotograph of the duodenal end of the hypertrophied mass. The normal pyloric sphincter is shown, entirely separated from the hypertrophied mass. (There is continuity between the pyloric and duodenal muscle in this specimen.) DG, Duodenal glands; S, Submucosa; DM, Duodenal muscle; P, Pyloric sphincter proper; C, Circular muscle; T, Hypertrophied mass.

the normal pyloric sphincter would be, however, and over an extent that would correspond with that of a normal sphincter, the normal circular fibres are thickened as they would be in a normal, though small, pyloric sphincter; and over this sphincteric thickening the superficial longitudinal layer almost disappears as such, as it does over the normal pyloric sphincter (*Fig. 354*). It is further to be noted that this normal pyloric sphincter makes no contribution to the hypertrophied mass, and is in fact separated from the duodenal end of the mass by a layer which is seen to be continuous with the submucosa and the muscularis mucosæ, and which, in the photograph, turns to the left over the proper sphincter (*Fig. 354*). These arrangements of the parts are represented diagrammatically in *Fig. 355*.

The gross arrangement of the parts in this case obviously resembles that

which is described in cases of 'congenital hypertrophic stenosis of the pylorus'; and we have been forced to the conclusion that the condition which is described here is a persistence into adult life of the infantile condition, with, probably, an increase in the hypertrophy—rather than an atrophy in the muscle of the other parts of the stomach—in the three years before admission. Such cases are extremely rare. It is now well recognized that chronic gastric disturbance in the adult may be due to obstruction at the pylorus which is not of the nature of reflex spasm or of cicatricial pyloritis, and several reports have been published of the anatomical findings in such cases as displayed at operation. The majority of these reports, for example those of Maylard,[1] Bastianelli, Cautley and Dent, and Landerer, describe conditions which are not comparable to those present in congenital hypertrophic stenosis—that is, there is in them a simple narrowing of the pyloric orifice either without or

FIG. 355.—A diagram of the arrangement of the muscle tissue : founded on *Figs.* 353 and 354.

more commonly with what is stated to be a simple ring-like hypertrophy of the pyloric sphincter ;* and this hypertrophy, when it is present, is unaccompanied by any thickening of the musculature of the pyloric canal as is characteristic of the infantile condition. The more extensive hypertrophy of the infantile condition was present in the case described by Oliver[2] in a man. age 51 years ; and its occurrence, and even fairly late appearance, was fully established in the post-mortem descriptions by Maier.[3]

This case, then, is one of persistent infantile hypertrophy ; and the arrangement of the parts, as represented in *Fig.* 355, seem to us to call for a re-examination of the pathological anatomy of 'infantile hypertrophy of the pylorus'—namely, that the hypertrophy does not affect the pyloric sphincter, which, indeed, rather remains undeveloped, but is only of the muscle wall of the whole length of the pyloric canal.† This thesis, which is supported by the study of the development of the parts, is the subject-matter of a following communication.

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* It must be extremely difficult at operation to decide what is and what is not a hypertrophied pyloric sphincter, apart altogether from the appearances of an oblique section of it ; for the limits of the anatomical normal are very wide.

† That is, of course, the pathological hypertrophy, for there is usually a functional hypertrophy of most of the stomach wall and also of the first part of the duodenum.

AN EXPERIMENTAL INQUIRY INTO THE ASSOCIATION BETWEEN GALL-STONES AND PRIMARY CANCER OF THE GALL-BLADDER.

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By some clinicians gall-stones are regarded as causal agents of biliary cancer. The foundations for this opinion are: (1) The frequency with which calculi are present in cases of primary carcinoma of the gall-bladder; and (2) The alleged experimental production of cancer by the insertion of foreign bodies into the gall-bladders of animals.

With the clinical evidence it is not the writer's purpose to deal, beyond remarking that the frequent association of cholelithiasis with carcinoma of the gall-bladder cannot in itself be regarded as a proof that either condition has caused the other. Both calculus and cancer may have developed in response to a common agency. Such a coincidence has been known to occur in the kidney, as the following example will show. Daels and Biltris[1] inserted a strip of collodion impregnated with radium sulphate into the kidney of a guinea-pig, which, twenty-two months later, was found to have (1) lithiasis, and (2) sarcoma of the kidney so treated. In this instance the sarcoma is to be attributed to the radium rather than to the calculus for several reasons, among which is the fact that Daels and Biltris were able in the same series of experiments to induce tumours by radium in other regions of the guinea-pig's body without the accompaniment of calculi.

In connection with the experimental side of the matter definite statements have been made to the effect that cancer of the gall-bladder has been produced by inserting gall-stones and other foreign bodies into its cavity, and such statements have received wide acceptance. The supposed experimental proofs will be discussed below.

LABORATORY EXPERIMENTS.

The first attempt to apply experimental tests in the laboratory was made by Kazama,[2] who introduced various foreign bodies into the gall-bladders of 98 guinea-pigs and produced cancer, as he believed, in 26 of these, 9 of the cases being accompanied by metastasis (Table I).

Leitch[3] undertook a repetition of Kazama's experiments, and inserted gall-stones into the gall-bladders of 25 guinea-pigs, pebbles into the gall-bladders of 5, and pilules of pitch into 5, and claimed to have brought about cancer in 8 of the animals within a year and a few days—5 of these cancers occurring within the first six months. Of the 8 cancers, 5 were caused by gall-stones, 2 by pebbles, and 1 by a pilule of pitch. Fifteen of the animals

were still alive a year after the operation, but their ultimate fate has not been recorded.

Nakamoto Joshiharu is mentioned by Genkin and Dmitruk[4] as having produced cancer of the guinea-pig's gall-bladder by the insertion of foreign bodies, but I have been unable to find a published report of his work.

Table I.—Results of Inserting Foreign Bodies into the Gall-bladders of Guinea-pigs (Kazama).

Nature of Material Inserted into Gall-bladder	Number of Guinea-pigs Used	Maximum Duration of Life after Operation	Cases in which Cancer was Produced	Cancer Cases with Metastasis
		Months		
Pebbles	30	$4\frac{1}{4}$	4	4
Suture material	23	3	6	1
Mucous membrane from gall-bladder	2	$1\frac{3}{4}$	2	1
Pityrol	16	$5\frac{1}{2}$	8	0
Lanolin	27	$4\frac{1}{2}$	6	3
Totals	98		26	9

Clemente[5] made similar experiments, inserting balls of tar mixed with cement 'the size of chick-peas' into the gall-bladders of 20 guinea-pigs. Nine of these animals died accidentally of 'insolation' three and a half months after the operation. The gall-bladders of these animals were thickened, hard, and adherent to the omentum and liver. Microscopical examination revealed the presence of neoplastic elements having a marked power of growth and invasion. The proliferative changes, he says, lead by progressive stages to adenoma, which afterwards gives place to atypical epithelial proliferation which in all probability can be interpreted as cancerous. All of these animals had increased in weight after the operation, the average increase at death being 122 grm. Professor Clemente[6] has with great kindness informed me of the fate of the survivors of this series and has generously given me permission to quote his observations. He says, " the results obtained in the 11 surviving guinea-pigs were not worth recording in an article. In fact, in most cases the gall-bladder was found to be intact, and there was no trace of the substance inserted. In the remaining cases there was true atrophy of the organ itself with retraction of the bile-ducts. Histological examination in these cases revealed sclerosis predominating on the epithelial proliferation. But even so, no characteristic lesions were found such as to recall those already described in the nine other guinea-pigs."

Delbet and Godard[7] inserted gall-stones from cancerous patients into the gall-bladder of 6 guinea-pigs and from non-cancerous cases into 10 guinea-pigs. Similar results followed in both groups. On examination at intervals varying from eight to nineteen months afterwards, the conditions found were fairly constant—namely, a thickening of the wall of the gall-bladder with

FIG. 356.—*Case* 1.

FIG. 357.—*Case* 2.

FIG. 358.—*Case* 3.

FIG. 359.—*Case 4.*

FIG. 360.—*Case 5.*

FIG. 361.—*Case 6.*

FIG. 362.—*Case 7.*

FIG. 363.—*Case 8.*

FIG. 364.—*Case 9.*

FIG. 365.—*Case 10.*

FIG. 366.—*Case 11.*

pronounced hyperplasia of the glandular elements, which formed cysts and tended to invade the liver.   The interpretation of the microscopical sections was not always easy, and changes which might be called 'precancerous' were observed.   In 2 of the cases the appearances were suggestive of cancer.   The authors criticize the former work of Kazama and Leitch, and do not regard their own work as having proved that cancer can be caused in the guinea-pig by the presence of gall-stones in the gall-bladder.

Petrov and Krotkina[8] introduced human gall-stones, either alone or together with paraffin and coal-tar, into the gall-bladders of 30 guinea-pigs. The experiment lasted twenty months, and in all the animals they found a

FIG. 367.—*Case 12.*

FIG. 368.—*Case 13.*

striking degree of epithelial hyperplasia.   Metaplasia was observed, and areas of atypical epithelial proliferation were seen surrounded by connective tissue and leucocytic infiltration.   The picture is described as that of precancer, though not of definite cancer, and they do not consider their experimental work to have proved the occurrence of cancer as a result of chronic irritation of the gall-bladder caused by the presence of foreign bodies.

Gioia[9] introduced various substances into the gall-bladders of guinea-pigs, including smooth pebbles, chips of pumice stone impregnated with coal-tar, and fragments of Chamberland filter which had been saturated with tar dissolved in xylol.   Independently of the nature of the foreign bodies employed there followed a vigorous fibro-myo-epithelial hyperplastic response ; moreover, the formed epithelial tissue spread into the adjacent structures, though not by infiltrating the lymphatics.   This hyperplastic response to injury took place with remarkable speed.   In one animal which died thirty-six
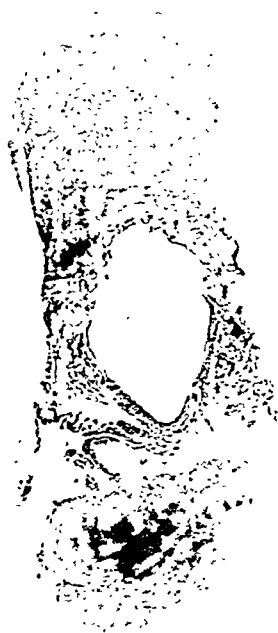
FIG. 369.—*Case* 14.
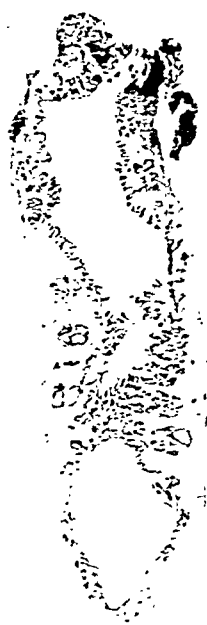
FIG. 370.—*Case* 15.

FIG. 371.—*Case* 16.

FIG. 372.—*Case* 17.

FIG. 373.—*Case* 18.

FIG. 374.—*Case* 19.
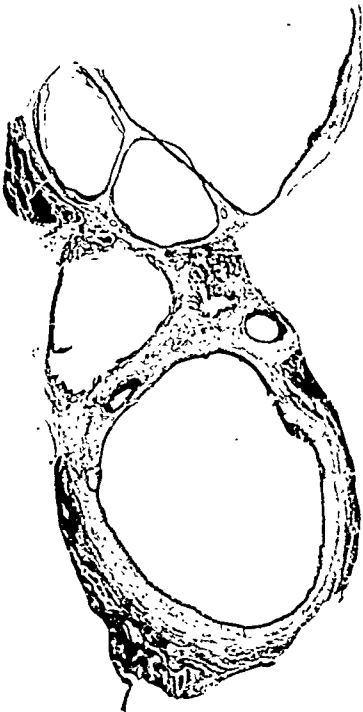
FIG. 375.—*Case* 20.

FIG. 376.—*Case* 21.

hours after the operation active epithelial proliferation with papillary formation was already in evidence. In none of his 17 cases could the death of the animal be attributed to the condition of the gall-bladder or of the adjacent organs. Although in some cases the microscopical appearances could be regarded as precancerous or as those of early cancer, in no case did metastasis or cancerous invasion of the neighbouring organs occur.

<div style="text-align:center">

FIG. 377.—*Case 22.*　　　　　　　FIG. 378.—*Case 23.*

</div>

Genkin and Dmitruk[1] experimented with 19 rabbits, introducing various foreign bodies (human gall-stones, pieces of pumice or of tile) into the gall-bladder. The foreign bodies were in some cases sterile and in others infected. The results were comparable with those obtained by the previous authors in guinea-pigs, except that in no case was there any indication of a malignant growth. Six of the animals survived four years after the initial operation and were then killed. The experimenters remark that as much papillomatous and adenomatous growth was found in the animals which died soon after the operation as in those which survived a long time.

Early in 1930, at the suggestion of the late Professor Leitch himself, the writer undertook to repeat this type of investigation, and biliary calculi were inserted into the gall-bladders of 47 guinea-pigs.

Technique.—A suitable area of the abdomen having been epilated, the guinea-pig was anæsthetized and the gall-bladder was exposed through a

paramedian incision made to the right of the xiphisternum. The gall-bladder having been opened between two clips applied to its wall, one or two calculi were inserted through the aperture so made. Closure was effected by rotating one clip around the other, so as to produce a twist of two turns, and then applying a fine catgut ligature. By this method no extraneous suture material was introduced into the viscus, nor did any inverted ridge of cut membrane remain within its cavity. The wounds healed well without leakage of bile, and the animals did not appear to suffer from any immediate ill effects after the operations.

FIG. 379.—*Case* 24.

The gall-stones used were of two kinds, and were derived from non-malignant human cases. Gall-stones 'A' were tetrahedral, faceted, clean cholesterol stones weighing between 50 and 100 mgrm. each. They were placed in methylated spirit prior to use. Gall-stones 'B' were smooth, irregularly faceted stones, which were opaque white on the surface and reddish-brown within, and were friable. They had been removed from a patient who had suffered from symptoms of cholelithiasis accompanied by thickening and fibrosis of the wall of the gall-bladder with some destruction of its mucous

FIG. 380.—*Case 25.*

FIG. 381.—*Case 26.*
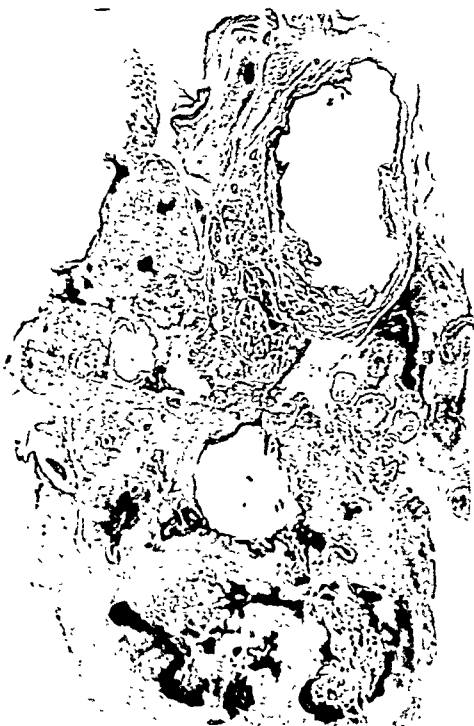
FIG. 382.—*Case 27.*
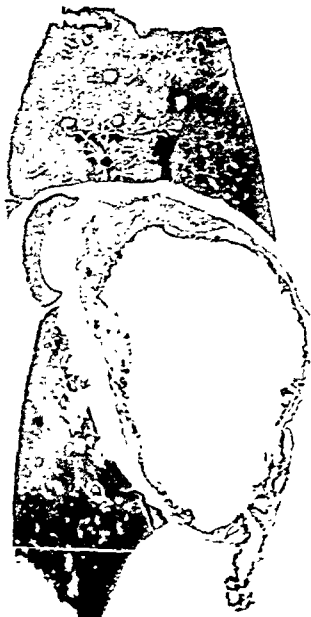
FIG. 283.—*Case 28.*

lining, no carcinoma being present. Usually only one of these stones was inserted, but this was sometimes broken into two or more fragments during insertion. These calculi were sterilized in the autoclave together with the dressings.

Results.—Some of the animals, owing to death in the early stages of the experiment, or because they died in the night and became too decomposed for satisfactory examination, or for other reasons, are not available for this

Table II.—Summary of the Author's Own Experiments.

	SEX	NUMBER OF STONES INSERTED	KIND OF STONE USED	LIFE AFTER OPERATION	CAUSE OF DEATH
				Weeks	
1	F.	1 (broken)	B	9	Not recorded
2	F.	1	B	11	Pneumonia
3	F.	1	B	17	Intestinal obstruction (adhesions)
4	F.	1	B	18	Not known (animal emaciated)
5	F.	1	B	27	Killed
6	M.	1	B	27	Killed
7	F.	2	B	27	Killed
8	F.	1	B	27	Killed
9	F.	1	A	27	Killed
10	F.	1	B	30	Killed
11	F.	2	A	30	Killed
12	F.	1	B	34	Pneumonia
13	F.	1	A	36	Killed
14	F.	1	A	36	Killed
15	M.	1	B	36	Killed
16	F.	1	B	36	Killed
17	F.	1	B	38	Killed
18	F.	1	B	46	Bronchopneumonia
19	M.	1 (broken)	B	54	Biliary sepsis
20	F.	1	A	61	Biliary obstruction
21	F.	1	A	73	Not recorded
22	F.	1	A	75	Pneumonia
23	F.	1	A	82	Nephritis
24	F.	1	A	91	Pneumonia
25	F.	1	A	93	Not recorded
26	F.	1	A	98	Not recorded
27	M.	1	B	98	Tuberculosis
28	F.	1	B	123	Nephritis
29	F.	1	A	128	Nephritis
30	F.	1	A	153	Nephritis
31	F.	1	A	154	Nephritis
32	F.	1	A	—	Still alive
33	M.	1	A	—	Still alive

report. There remain 33 whose after-history can be accurately followed (*Table II*). Not one of these animals has succumbed to cancer, and no cancer has been found microscopically. Two of the animals are still alive and well nearly three years after introduction of the gall-stones. In every case examined after death—31 in all—a condition of hyperplasia has been present in the gall-bladder. Although varying as between one individual and another in minor characteristics and degrees, the changes found conform

to those described so admirably in this JOURNAL by King and MacCallum[10] as occurring in cases of human cholelithiasis. under the title of " Cholecystitis Glandularis Proliferans (Cystica) ". In this respect my results agree with those of every other experimenter (*Figs.* 356–384).

FIG. 384.—*Case* 29.

The changes may be expressed as follows. The gall-bladder. which in the guinea-pig is normally almost free from the liver (*Fig.* 385). becomes adherent to it by newly formed connective tissue so as to appear in some instances completely embedded (*see Figs.* 358. 365. 369). The resultant mass is hard to the touch. often shows rounded bosses. is dead white. and without sharply defined outlines. Owing to these features the condition may readily be mistaken macroscopically for a malignant growth. Such remarks can be applied equally to man. *Fig.* 386 is a photograph of this benign condition as represented by a specimen of human origin in the museum of the Cancer Hospital. and it shows well how closely the condition may simulate cancer when viewed with the naked eye. In *Fig.* 387. which is taken from a microscope slide of this specimen. the condition will be seen to correspond with

that which occurs in guinea-pigs under the experimental conditions now being discussed. Microscopical examination of the lesions produced in the animals reveals extensive proliferation of the fibrous, muscular, and glandular elements of the gall-bladder. The hyperplastic epithelium projects into the cavity of the viscus to form papillomata (*see Figs.* 373, 376), grows outward through the muscular bundles, forming subperitoneal adenomata where the gall-bladder is free (*Figs.* 378, 388), and invading the liver where the two organs are joined together by newly formed connective tissue (*see Figs.* 358, 365). In these protrusions cystic dilatations are very frequent and the epithelium is often atypical (*Fig.* 389). Along the surface where the liver and the newly formed

FIG. 385.—Gall-bladder from a normal guinea-pig.

connective tissue of the gall-bladder come into contact there are seen many small bile-ducts (*Figs.* 390, 394) in which the arrangement of epithelium may be disordered and suggestive of an early malignant change.

DISCUSSION.

The general character of the results obtained in all the experiments by the different observers mentioned above is uniform—that is to say, the introduction of a foreign body into the gall-bladder of a guinea-pig or a rabbit produces at a very early stage a rapid and extensive proliferation of the various histological elements composing the affected viscus. Moreover, this proliferation is accompanied by a penetration into the contiguous structures— for example, the liver and adherent omentum—of the newly formed glandular

and other elements derived from the gall-bladder (*see Fig.* 392).   This invasive
process has led to varying interpretations by individual workers, some having
accepted it—especially when accompanied by the development of atypical
epithelium—as evidence of malignancy, while others have regarded this kind

Fig. 386.—Cholecystitis proliferans in man. Photograph from specimen No. 1322 in
the Museum of the Cancer Hospital (Free), London.

of invasion as compatible with innocence.   Some have avoided a final decision
by referring to the conditions as precancerous.   As will be seen below, the
writer does not believe that the microscopical evidence hitherto brought
forward to support the view that cancer has been produced artificially by

gall-stones is impeccable. However that may be, the diagnosis of cancer in experimental work with animals requires further proofs than those supplied by the microscope. The tumour should infiltrate and destroy the neighbouring structures, it should be amenable to transfer by autografts or heterografts, it should form metastases, and. unless treated, it should progress so as to kill the host. Not all these tests are essential to a diagnosis of malignancy, but at least some of them should be fulfilled. In the experimental work now under consideration not one of them can be used to substantiate the diagnosis of cancer. Even the protrusion of newly formed glandular elements from the inflamed gall-bladder into the liver (*see Fig.* 392), though described as
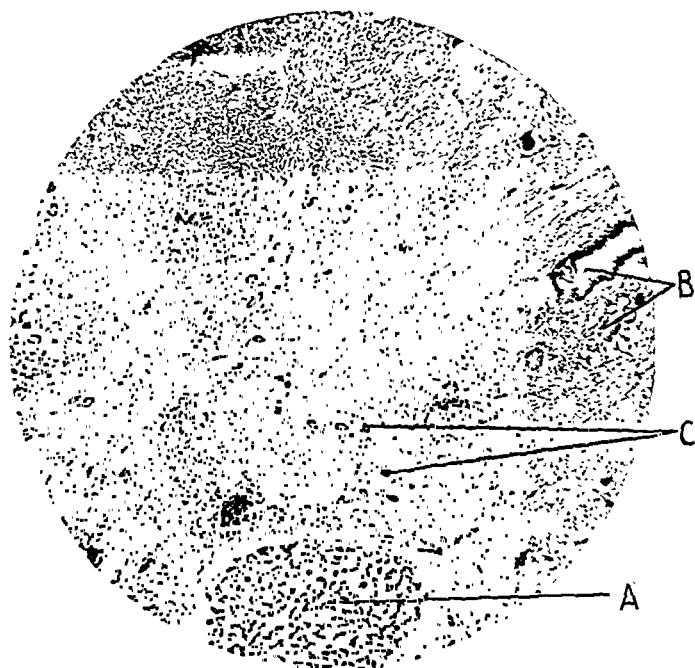
Fig. 387.—Microscope section taken from specimen illustrated in *Fig.* 386. The main ground is granulomatous. An isolated group of liver cells (A) is shown in addition to adenomatous tissue (B) derived from the gall-bladder, and numerous small ducts (C) probably originating from the liver.

'invasion', differs in character from the infiltrative and destructive ingrowth of malignant disease. It appears to be preceded by a fibroblastic development, the protrusion of glandular epithelium being confined to the granulation tissue.

The absence of any successful grafting of the tumours in the experiments described above is significant. Kazama seems to have been the only experimenter to make such an attempt, and his efforts did not succeed. The failure or omission of this important test appears inexplicable unless one is to assume either that the tumours were innocent or that neoplastic growth of sufficient bulk was not available—a lack which in itself might cast a doubt on the diagnosis. Kazama is the sole inquirer amongst those quoted who states

that he has seen metastases. Unfortunately his paper is not illustrated. In one case he claims to have produced cancer with metastasis within the astonishingly brief period of seven weeks.

Concerning the lethal nature of the condition produced, it is noteworthy that among the 268 animals into whose gall-bladders foreign bodies have been introduced by the various workers (*Table III*) in the foregoing experiments

Table III.—RESULTS OF INSERTING FOREIGN BODIES INTO THE GALL-BLADDERS OF 268 ANIMALS.

AUTHOR AND YEAR OF PUBLICATION	ANIMAL USED, AND NUMBER	FOREIGN BODIES USED	MAXIMUM DURATION OF LIFE AFTER OPERATION	NUMBER FREE FROM CANCER	NUMBER WITH CANCER	CANCER WITH METASTASIS	SURVIVORS IN WHICH ULTIMATE RESULTS HAVE NOT BEEN RECORDED
			Months				
Kazama, 1922	Guinea-pig 98	Gall-stones Suture material Mucous membrane Pityrol Lanolin	5½	72	26	9	—
Leitch, 1924	Guinea-pig 35	Gall-stones Pebbles Pitch pills	12	27	8	0	15
Clemente, 1927	Guinea-pig 20	Pills of cement and tar	3½	20	0	0	0
Delbet and Godard, 1928	Guinea-pig 16	Gall-stones	19	14	2 ?*	0	0
Petrov and Krotkina, 1928	Guinea-pig 30	Gall-stones Paraffin Coal tar	20	30	0	0	0
Gioia, 1929	Guinea-pig 17	Pebbles Pumice and tar	23	?	? †	0	0
Genkin and Dmitruk, 1932	Rabbit 19	Gall-stones Pumice Tile fragments	48	19	0	0	0
Burrows, 1932	Guinea-pig 33	Gall-stones	38½	33	0	0	2‡

° In two cases there were appearances suggestive of cancer. + In some cases the microscopical changes could be regarded as precancerous or as early cancer. ‡ These animals are still alive.

not one is recorded to have died of cancer. If, as stated in Kazama's and Leitch's papers, malignant tumours were in fact brought about within a period of two to six months, it is difficult to explain why none of the animals died of the disease, seeing that many of them survived after operation for periods much longer than six months. Apparently it must be assumed either that the diagnosis of cancer was at fault, or else that only those animals having cancer already happened at an early stage to succumb to some intercurrent

condition or, though seeming to be well, to have been sacrificed deliberately. The latter alternatives are so improbable as to be hardly acceptable.

From these arguments it is clear that the only basis for supposing cancer to have been produced by inserting foreign bodies into the gall-bladder, is the appearance seen in certain microscope sections taken from animals whose deaths could not in any way be attributed to the presence of cancer. It remains to be seen how far these sections are to be relied upon. Creighton[11] called into question the diagnosis of malignancy as shown by the illustrations of Leitch's paper, and the present writer has not yet seen a microscope section or an illustration which convinced him that the condition of the gall-bladder brought about by the experimental introduction of foreign bodies was in fact
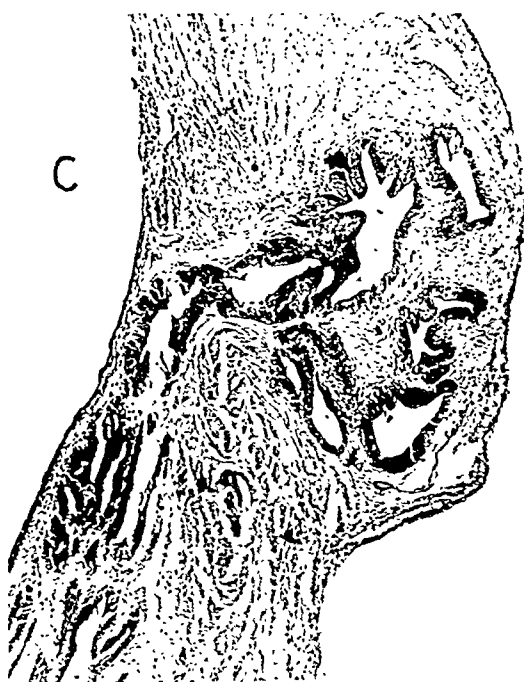
FIG. 388.—*Case 9.* Part of the wall of the gall-bladder. A glandular protrusion has grown through the hypertrophied muscle to form a subperitoneal adenoma. c, Cavity of gall-bladder.

malignant. Certain fallacies occur in the particular circumstances under consideration. The first is the ingress of glandular protrusions derived from the gall-bladder into the liver and other adherent tissues. This 'invasion', as remarked above, always appears to be preceded by freshly formed fibro-blastic tissue to the region of which the glandular proliferation is confined (*see Fig.* 392). In this respect the condition differs essentially from a malignant invasion. The ingrowths of fibroblastic tissue, whether containing epithelial cells or not, may cause the isolation and destruction of small groups of hepatic cells (*Figs.* 387, 393). The presence of such isolated masses of parenchyma-tous cells in itself seems to have been regarded as evidence of malignancy

FIG. 389.—*Case* 12. Shows general hyperplasia of wall of gall-bladder with formation of cystic adenoma.
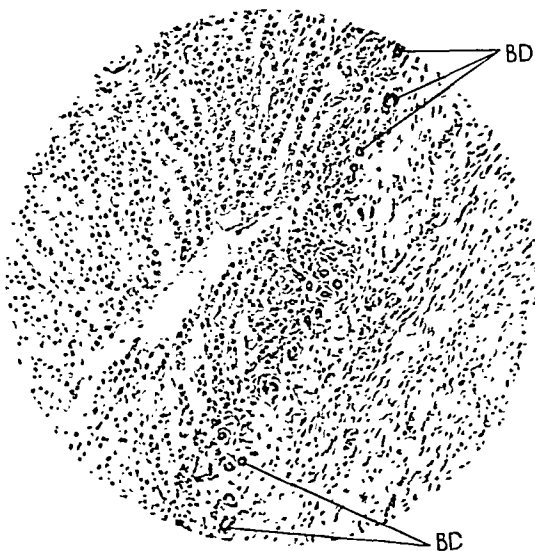
FIG. 390.—*Case* 26. Illustrates proliferation of small bile-ducts (BD).

(see Leitch). However, this permeating or, as it might be termed, infiltrating fibroblastic response to irritation of the gall-bladder can hardly be mistaken for anything but an innocent condition. Any misunderstanding that has

FIGS. 391, 392.—*Case* 3. Illustrate an ingrowth into the liver of fibroblastic tissue containing glandular epithelium derived from the gall-bladder. In *Fig.* 391 a so-called 'islet' is shown, which serial sections prove (*Fig.* 392) to be continuous with the gall-bladder.

arisen in this respect must be attributed to the fact, already mentioned, that proliferating glandular epithelium derived from the gall-bladder is apt to extend along these fibroblastic processes and so to form simple protrusions

or compound ramifications of adenomatous looking tissue within the body of the liver. This intruded glandular tissue frequently becomes cystic. In single microscope slides 'islets' of such proliferating glandular tissue may be seen in the liver substance accompanied, it may be, by so-called cysts. Such islets have been mistaken for deposits of adenocarcinoma, though serial sections demonstrate that they are in reality adenomatous protrusions from the gall-bladder with which they are in direct continuity (*Figs.* 391, 392).

Another source of error is the formation by the liver of new bile-ducts in response to irritation. The phenomenon is recognized in connection with acute yellow atrophy and hepatic cirrhosis, and can be seen in many of the microscope sections of a chronically inflamed gall-bladder in contact with the liver (*Fig.* 390). These ducts, originating from the liver, and becoming
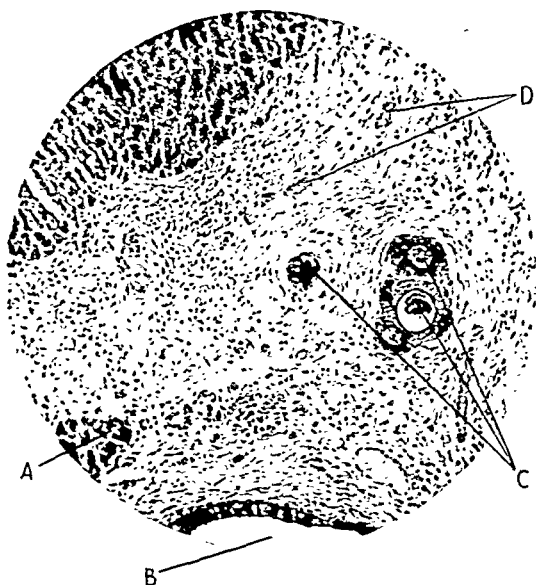
Fig. 393.—*Case* 10. Shows an isolated group of liver cells (A), adenomatous cyst (B), proliferated glandular epithelium from the gall-bladder (C), and ill-formed bile-ducts (D) derived either from the liver or the gall-bladder.

associated with proliferating glandular epithelium from the gall-bladder which also is sprouting into the newly formed fibroblastic tissue in and about the liver, may be misinterpreted as derivatives of the gall-bladder invading the liver.

In *Fig.* 394 is shown a thin adhesion between the gall-bladder and liver (*Case* 25, *Table II*). At the hepatic end of the adhesion a small area of granulation tissue is seen encroaching on the parenchyma of the liver, and in this granulation tissue, close to the hepatic cells, is seen a small mass of proliferating bile-duct epithelium, presumably derived from the liver. Serial sections taken through a considerable extent of this adhesion fail to show a direct connection between these new bile-ducts and the glandular elements of the gall-bladder in the neighbourhood. The absence of any adenomatous

encroachment from the gall-bladder in the neighbourhood of the adhesion to complicate the picture in this specimen renders the hepatic origin of these newly formed ducts almost if not quite convincing.

To the foregoing criticisms of past experimental work some supplementary comments of a more general nature may be added. Among them is the curiously short period in which the supposed experimental cancers arose. Thus Kazama claimed to have produced two cases of carcinoma, one of them with metastasis, within a period of seven weeks, and the maximum duration of his experiments seems to have been five and a half months (see Table I); and Leitch reported the production of carcinoma in five cases within six months. These periods are suspiciously short for the induction of cancer in the guinea-pig by artificial means.

Fig. 394.—Case 25. A thin adhesion connects the gall-bladder with the liver. At the hepatic end of the adhesion is a small area of granulation tissue, in which, close to the liver, are numerous small bile-ducts (see text).

Another point is this : all the experimenters to whom reference has been made above have obtained the same results whatever foreign body they have used, whether gall-stones or other material—that is to say, the outcome has been the consequence of a non-specific irritation. But it is recognized that cancer is an infrequent result of non-specific irritation as applied to laboratory animals other than the guinea-pig.

The experiments on which this paper has been founded call to mind the somewhat similar procedure carried out by Pack and Buzzanca,[12] who inserted sterile pebbles into the renal pelves of rabbits and thereby produced epithelial hyperplasia and papillomatosis. No malignant disease occurred, but the

longest post-operative survival was only 177 days, and it is conceivable that cancer might have supervened if the rabbits had lived longer.

In conclusion, it appears that laboratory experiments up to this date have failed to establish a causal relationship between the presence of calculi in the gall-bladder and the occurrence of cancer in this organ. Further experiments are in progress and the results will be reported.

SUMMARY.

1. Gall-stones inserted into the gall-bladders of 33 guinea-pigs did not produce cancer.

2. Previous experiments by other investigators have been analysed in view of these results, and are held to be in accord with them.*

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* Since this paper was written Petrov and Krotkina[13] have recorded the results of some further experiments. They used 19 young guinea-pigs, inserting glass tubes containing radium into the gall-bladders of 12, and glass tubes without radium into the gall-bladders of 7. They state that after periods of 136 and 158 weeks respectively carcinoma was found in 2 of the animals which had been treated with radium, while of the guinea-pigs treated with plain glass tubes, adenocarcinoma with metastases occurred in 2—in the 66th and 166th weeks of the experiment.

POLYORCHIDISM.

By R. HODGSON BOGGON,

FROM THE SURGICAL UNIT, ST. THOMAS'S HOSPITAL.

POLYORCHIDISM is a condition which always has excited considerable interest from legendary to scientific times.   In the old days a large number of cases were described, and those blessed with this numerical curiosity were thought to be greatly favoured and possessed of supervirility.   Lucas-Championnière discoursed on the subject in a lecture in 1900 and stated that Ambrose Paré, as well as many other surgeons of that period, believed multiple testes to be not infrequent.   Cases of three, four, or five testes were recorded, and authors appeared to compete one with another in the number of testes in each case described, and in fact a new nomenclature was evolved.   Arnaud in the eighteenth century apparently believed most unusual stories.   He quoted the case of a man endowed with polyorchidism who practised coitus up to the age of 125 years and other instances of men able to repeat the act twenty-five to thirty times in one night.   When reduced to fact the only authentic example of the condition in early days is confined to a post-mortem by Blasius in the seventeenth century on a patient with three testes.

With the advent of scientific times and more critical investigation the number of cases has rapidly diminished.   Only those should be accepted in which the 'supernumerary testis' is submitted to histological examination, for there are many other conditions, such as a cyst of the cord or a floating epididymis, which may be mistaken for it.

Edington and Blacklock in 1928, in a careful investigation of the literature, were able to find only eight cases (to which they added one), that were submitted to microscopical examination.   King in 1931 reported an additional example and mentioned also a post-mortem case of Ahlfeld's.   In view, therefore, of the rarity of the condition it is desirable to put this, the twelfth, case on record, especially as it was associated with recurrent torsion of one of the testes.   The following are the notes of the case :—

CASE REPORT.

HISTORY.—Male, age 23.   Baker by trade.   Married and has one child.   In 1928 he was operated upon successfully for a thyroglossal fistula.   No other relevant previous history.   For the five years previous to admission the patient experienced temporary swelling and acute pain in the left side of his scrotum.   These attacks occurred about twice a year and lasted several hours.   The last attack was eight months before admission.   Typically the attack occurred suddenly without any warning, and the patient was unable to say what brought it on.   During the attacks the pain was very severe and radiated up into the left groin, the left testicle at the same time becoming swollen and very tender.   The termination of an attack was fairly abrupt and the swelling soon subsided.

ON EXAMINATION.—The patient is a man of sallow complexion and poor physique.   On his neck is the scar of the healed thyroglossal fistula operation.   On

examining his scrotal region it was noticed that the right testicle hung lower than the left and appeared on palpation to be of normal size and in a normal position. In the left side of the scrotum, however, there were two small ovoid bodies, one about twice the size of the other. The larger of the two lay at the bottom of the scrotum and was about 1 in. in diameter. It gave no testicular sensation on pressure. One and a half inches above, and placed somewhat antero-laterally, lay the second swelling, which did not appear to be so definitely round as the lower one and was similarly devoid of testicular sensation. Between the two, and joining them, could be palpated a soft tape-like structure, and above, leading up to and through the external abdominal ring, the vas with its accompanying structures. The provisional diagnosis was imperfectly descended testicle with separation of the epididymis from the body of the testis, the symptoms being due to recurrent torsion.

OPERATION.—Under general anæsthesia operation was carried out on July 15, 1931, by Mr. Maybury. A left inguinal incision was made and the inguinal canal
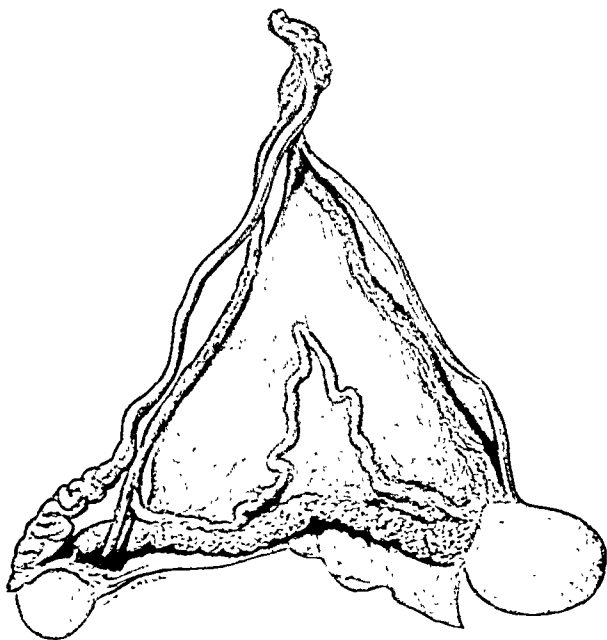
FIG. 395.—The specimen after dissection.

opened by cutting through the external oblique aponeurosis. The spermatic cord was pulled upon and the left scrotal contents were delivered into the wound. It was then found that the two solid structures had the appearance of two testicles enveloped in tunica vaginalis. A band of tissue ran down from the external ring to both, and owing to this it was thought that the vas divided. When the tissue was dissected at a later date it was found that the band to the larger testis consisted only of vessels. In view of the recurrent acute symptoms for which the operation was being performed and the fact that the testicles were small and imperfectly developed (as evidenced by the lack of testicular sensation), it was considered advisable to remove them. As there was no evidence of a hernial sac, the cord was ligatured and divided by the internal abdominal ring and the testicles were removed. There was no evidence of a gubernaculum. The patient made an uneventful recovery and was discharged on July 22.

MACROSCOPIC DESCRIPTION.—The tissue removed at operation was preserved and dissected at a later date (*Fig.* 395). It consisted of two ovoid bodies of unequal

size, both enveloped in tunica vaginalis, which extended up to near the external abdominal ring. Lying at the lower end of the mass was a small quantity of fibro-fatty tissue, which might be the remains of the gubernaculum, for otherwise there was no evidence of this structure. The two ovoid bodies on section proved to be testicles. The smaller of the two measured 1·5 × 1·25 cm. Running down to it from the external abdominal ring was the cord consisting of the vas together with its vessels. The vas was tortuous near this smaller testis, and, where it was con-nected to its outer side, became very narrowed and coiled like an epididymis.

The larger testis lay about 5 cm. below the smaller and somewhat to the inner side. It measured 2·5 × 2 cm., and was connected by a structure which on dissec-tion had the appearance of an epididymis. This epididymis was 7·5 cm. long with a width of 5 to 6 mm. and a thickness of 4 mm., and joined the larger testis at its upper pole. The tunica had to be carefully dissected away to reveal its structure.

FIG. 396.—Microphotograph of a section of the larger testis.

The vessels ran down in relation with the vas, and on reaching the smaller testis gave off a branch which ran at first along the upper border of the epididymis, then for a short course upwards in the scrotum, again to the epididymis, and reached the larger testis. From the upper border of this testis another mesh of vessels ran upwards and somewhat posteriorly to join the cord just below the external abdominal ring.

MICROSCOPIC DESCRIPTION.—The larger testis is surrounded by a thick fibrous capsule (*Fig.* 396). Internal to this there is evidence of massive necrosis followed by organization. The tubules have to a large extent disappeared and are replaced by fibrous tissue. Here and there remain a few tubules which show no evidence of spermatogenesis. The interstitial cells of Leydig can be seen clearly and the cells of Sertoli are just demonstrable (*Fig.* 397). In other areas the fast disappearing tubules are seen as indistinct outlines in a matrix of fine fibrous tissue. New capillary vessels are visible and the old vessels show hyaline degeneration of their

walls. Small areas of old hæmorrhage with pigment-granules are scattered about in the fibrous tissue.
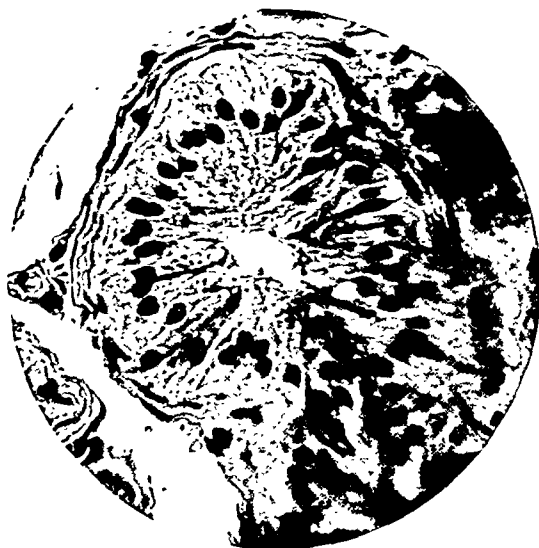
FIG. 397.—High-power view of a tubule from *Fig.* 396.

The small testis has a well-marked fibrous capsule. It contains a large number of tubules, many more than in the larger testis, but of a poorly developed type,

FIG. 398.—Microphotograph of a section of the smaller testis.

and in some places there is evidence of degeneration (*Fig.* 398). Spermatogenesis was not taking place, although the tubule-cells have stained more darkly than

normal (*Fig.* 399). The interstitial tissue contains in addition to fibrous tissue many cells of Leydig. The scattered blood-vessels are normal in appearance.
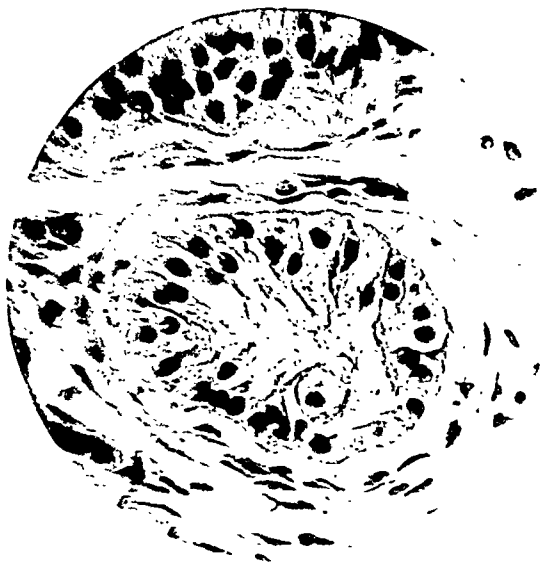
FIG. 399.—High-power view of a tubule from *Fig.* 398.

The elongated epididymis between the two testicles consists of loosely packed tubules (*Figs.* 400, 401). These tubules are lined by two layers of epithelium, the nuclei of which stain a great deal more darkly than normal. The cells of the

FIG. 400.—Microphotograph of a section of the epididymis in relation to the larger testis.

basal layer are small, dark, and cubical, while those of the other layer are columnar and less deeply staining with basal nuclei. The stereocilia are normal and the

lumen is completely empty of spermatozoa—good evidence of the non-activity of the larger testis. In the intertubular space are fibroblasts and fibrous tissue, but the latter is considerably denser immediately adjacent to the tubules than normal. The blood-vessels are dilated. Deposits of fat in the fibrous tissue (shown up by staining with Scharlach R.) indicate a degenerative process. The vas is normal in its histological structure (*Fig* 402).

Fig. 401.—Microphotograph of a section of the epididymis in relation to the smaller testis.

DISCUSSION.

The points of interest in this condition, besides its rarity. relate to the factors concerned in the production of the abnormality, and its association with torsion. Arbuthnot Lane in 1895 was the first to record an example of polyorchidism that was proved by operation and histological examination. although Ahlfeld's specimen found at post-mortem is of earlier date.

The specimen here described appears to be identical in principle with those described by Lossen. Edington. and King—namely, two testes with a single vas and an epididymis stretched out in such a way as to serve them both. In Lossen's and Edington's cases, however, the vas arose from the epididymal structure in relation to the larger testis.

In order to explain the mode of production of the two testicular bodies. it is necessary to outline briefly the normal embryological changes. The testis develops from the genital ridge, a longitudinal thickened fold on the inner surface of the Wolffian body (*Fig.* 403. A). The germinal epithelium of the middle third of this ridge invades the subjacent mesoderm to form the sex cords and ultimately the seminiferous tubules. The rete cords most

probably arise from the germinal epithelium of the anterior portion of the genital ridge, while the vasa efferentia are derived from the anterior part of the mesonephros, or Wolffian body (*Fig.* 403, B). The epididymis and vas deferens arise from the Wolffian duct. During development the anterior seminiferous tubules disappear and new tubules are formed, more caudally. In this particular form of double testis it is suggested that this disappearance of tubules is at fault, so that they disappear in the middle of the genital ridge only, while anteriorly they persist (*Fig.* 403, C). This would explain the common epididymis and vasa efferentia to both testes. King states that in his opinion the abnormality is due to transverse division of the genital ridge. In his case there was a well-marked gubernaculum, which on section showed smooth muscle and fibrous tissue, attached to the upper and larger testis,
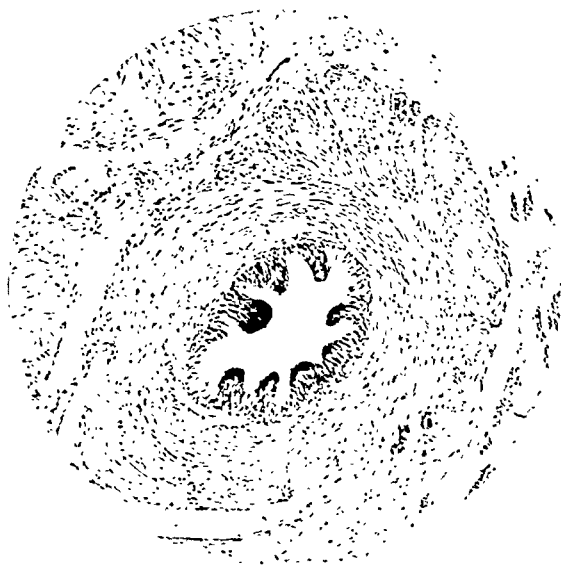
FIG. 402.—Microphotograph of a section of the vas deferens.

which arose from the cephalic end of the genital ridge. He endeavours at some length to explain this method of descent, and I have similarly attempted to show by means of diagrams how descent may have occurred in the case reported here, although there was no evidence, as far as can be ascertained, of a definite gubernaculum. It is inferred that in the developmental position the larger testis was in the uppermost or cephalic position and to it the termination of the epididymis is attached. The artery to this testis should be the shorter, as the latter is nearer in its origin from the aorta. In the descent into the scrotum (*Fig.* 404, A) the larger testis, having the gubernacular attachment, descends so as to become the inferior organ (*Fig.* 404, B).

Spermatogensis is described as occurring in at least six of the cases in the literature, and from the accounts it seems probable that this degree of development depends partly on the size of the testis and partly on the distance travelled from the intra-abdominal position. In this specimen there

was no evidence in spite of the size and descent of the organs. This may be explained in the larger testis by the recurrent twistings to which it was subjected and which interfered with its normal physiological activity. In the rest of the records there is either no mention of the functional condition, or spermatogensis was not present.

In some cases the abnormality was of a different type, the supernumerary organ having a separate vas (Lane, Haas), while in several others (Lerat, Mariotti, Lecène) a separate cord was described, which may or may not have included a vas. It is difficult to be sure on this point without careful dissection, as in the case described here there appeared at first sight to be two vasa, one of which was shown later to consist of a leash of vessels only.
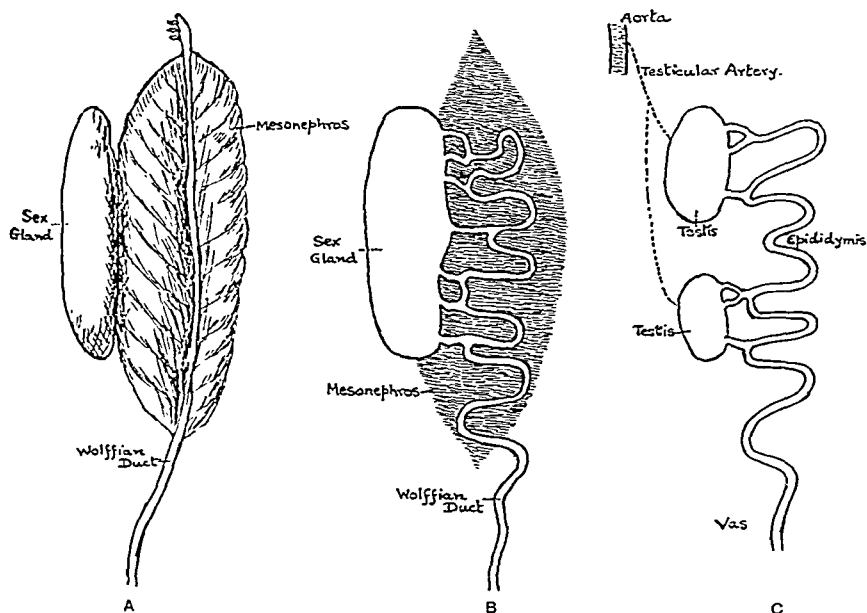
Fig. 403.—A, Diagram illustrating the position of the undifferentiated sex gland; B, Diagram showing the formation of the body of the testes, vasa efferentia, epididymis, and vas deferens; C, The formation of the two bodies of the testes.

A duplication of the vas appears to be associated with a more marked cleavage of structures in the region of the mesonephros during the process of development.

It is convenient to mention here what is termed by Edington and Blacklock 'pseudo-duplication', as it can be easily confused with true polyorchidism. Cases are reported by Marsh, Banks, and Lowe. In Marsh's specimen two testes were present in the left side of the scrotum, while the right side was empty. Two vasa ran from the testes and joined to form a common duct, while the right spermatic artery, arising normally, crossed the mid-line to supply one of the testes, after joining the left artery.

The preponderance of left-sided cases is striking, for of the 12 recorded no fewer than 9 were on this side. The explanation is hard to find.

The occurrence of recurrent torsion is interesting, and the evidence for it does not leave much room for doubt. The recurrent and sudden attacks of acute pain and swelling were the cause of the man's applying for treat-ment, and it is more than likely that if these symptoms had been absent, this specimen would never have been obtained. The necrotic destruction of the tubules seen on section, with the evidence of organization, indicate a history of some length. In the cases subjected to microscopy there is not one record of torsion, although Holder's example might be considered. His patient was admitted with four days' acute pain and swelling in the left inguinal region. A diagnosis of suppurative adenitis was given and an incision made. A testis was found which, on being examined microscopically, showed
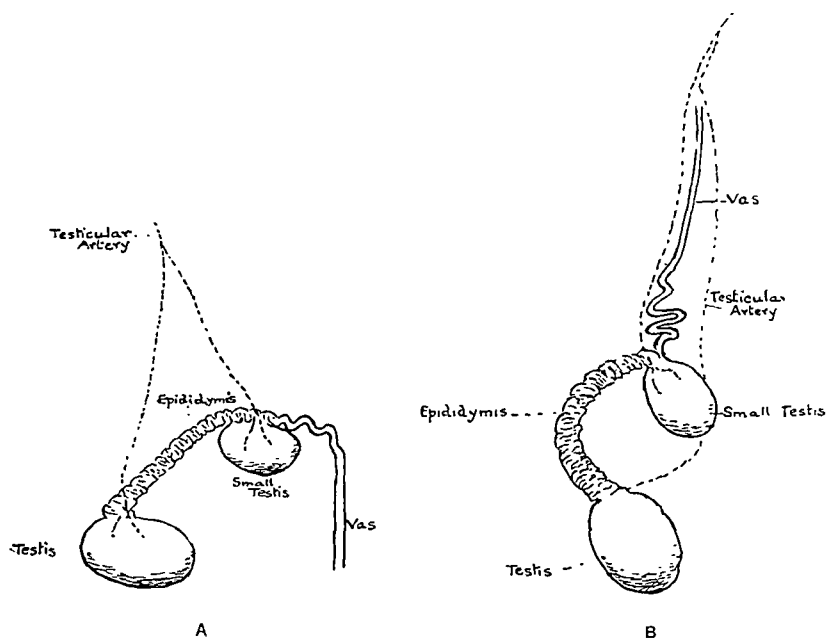
FIG. 404.—A, The early descent of the organs; B, The final descent into the scrotum.

active spermatogenesis with a condition of acute inflammation. From these data it is probable that the correct diagnosis was acute inflammatory orchitis. Coudert and Derocque described a case which can hardly be included in this series, as no description of the section was published. This patient was a child of seven months, admitted with the symptoms of a strangulated inguinal hernia, and was operated upon by Derocque. At operation in the inguinal region he found a tumour exactly like an acute torsion of testis, while below in the scrotum lay a normal testis. The twisted organ arose from the cord by a pedicle and appeared to have an epididymis. If this was a case of polyorchidism, then it is the only other example of such an anomaly associated with torsion.

The causation of torsion is still largely speculative, but the well-recognized etiological factors of imperfect descent and formation were present in this

case. and the possible factor of complete envelopment by tunica vaginalis as put forward by Muschat may also have assisted.

I am indebted to the late Sir Percy Sargent and Mr. Maybury not only for permission to publish the records of this case but also for their kind advice ; to Professor le Gros Clark and Dr. D. C. L. Derry for generous assistance in the anatomical and pathological details respectively ; and to Mr. F. E. Bloss for so ably preparing the microphotographs.

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CARCINOMA OF THE MALE URETHRA.

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THE case of carcinoma of the male urethra reported here is worthy of record not only because of the rarity of the condition, but also because the growth was situated in the fossa navicularis, an uncommon position. In addition, the extremely early diagnosis and treatment of the patient are of interest. Up to 1929 Huggins and Curtis were able to collect only 110 cases from the records in which there was but little doubt of the diagnosis. Since then a few more have been put on record, mostly in the foreign literature.

The first example of carcinoma of the urethra to be published was described by Thiaudière in 1834, but subsequently Kaufman has thrown some doubt on the diagnosis. Robb notes the interesting fact that up to 1928 there was no specimen of this condition in the museums of either the Royal College of Surgeons of London or Edinburgh. No doubt this is due in part to the fact that early cases are rarely seen, but it is largely because the majority of urethral carcinomata originate in the membranous and bulbous urethra, where invasion of surrounding tissues prevents removal in the form of a specimen.

The following are the notes of a case admitted to St. Thomas's Hospital under the care of the late Sir Percy Sargent, to whom I am indebted for permission to publish these details.

HISTORY.—Male, age 60. In his youth he had suffered from both gonorrhœa and syphilis, but did not develop a urethral stricture. For years he had suffered from epileptic fits, which were controlled by luminal. Three months before admission he noticed a slight bloody discharge from the external urinary meatus, and this drew his attention to a small nodule on the under surface of his penis.

ON EXAMINATION.—A hard nodule about the size of a split pea was felt on the under surface of the glans penis in the line of the urethra and about one inch from the external meatus. The tumour was subcutaneous, and when squeezed a drop of bloody discharge exuded from the urinary meatus. In the right inguinal region two rubbery and movable glands were felt. The general condition of the patient was poor. A provisional diagnosis of carcinoma was made and it was decided to explore the urethra in order to verify this.

OPERATION (Feb. 17, 1932).—An operation was carried out under general anæsthesia. The floor of the urethra was split open from the external meatus to the site of the tumour, which consisted of a small ulcer with an indurated base and edge. This was considered to be malignant. In view of the uncertainty of the results of treatment by radium, it was decided that a conservative

amputation should be performed. The penis was therefore removed from in front of the scrotum, using a dorsal flap. A catheter was inserted and removed after twenty-four hours. The patient made an uninterrupted recovery and was discharged on March 5. As the glands in the right inguinal region were small and movable, and in view of the general state of the patient, no attempt was made to remove them at this time.

FIG. 405.— The amputated penis, showing the growth from the ventral aspect.

NAKED-EYE APPEARANCES.—The specimen (*Fig.* 405) shows the amputated penis from the ventral aspect, with the urethral floor split open. The growth can be seen arising from the roof of the fossa navicularis and running round the lateral walls, but not to a sufficient extent to cause, at this stage, a urethral stricture. The resultant ulcer has a surface with a red granular appearance, and an edge that is raised and everted. Deep to it the growth invades the substance of the glans to a slight extent. Between the growth and the external urinary meatus there is quite definitely an area of normal urethra.

MICROSCOPIC APPEARANCES.—The following report of the microscopic examination was provided for me by Dr. D. C. L. Derry, the Pathologist to the Surgical Unit, to whom I take this opportunity of expressing my thanks :

" On section the tumour shows cords of typical epithelial cells invading the lamina propria of the glans. These cells have large round or oval vesicular

FIG. 406.—Low-power microphotograph of the malignant tissue.
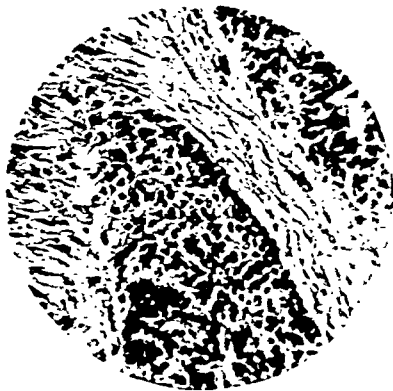
FIG. 407.—High-power microphotograph of a portion of *Fig.* 406.

nuclei, with a large eosinophilic nucleolus, and pale-staining granular cytoplasm. A few mitotic figures are present, and the usual round-celled

infiltration surrounds the growth. There are no 'prickle-cells' or 'epithelial pearls' among the cords of tumour cells. Diagnosis : squamous-celled carcinoma of the transitional-cell type." (*Figs.* 406, 407.)

DISCUSSION.

The age incidence of this condition has wide variation. The youngest patient was a youth of 18 years, described by Paton, whilst at the other extreme is a nonagenarian. The disease, however, appears to be commonest between the sixth and seventh decades. The etiological factors, as ascertained from the literature, are a previous history of gonorrhœa, trauma from the passage of sounds or other causes, and chronic urinary infection. Gonorrhœa easily heads the list, usually associated with stricture. In one of Robb's cases it is interesting to note that sounds were passed on 477 occasions, a particularly inquisitorial method of producing chronic irritation. Kretschner associated a case with the irritation produced by the injection of Hartzell's solution.

With regard to the site, there is a great affinity for the perineal urethra. Diehl, in reviewing 61 cases, found that 33 were in the membranous urethra, 26 in the bulbous urethra, and only 2 in the fossa navicularis. Other authors report much the same proportions. This common position is to be expected, as the predisposing factors of gonorrhœa and stricture make their mark mostly in this region.

The outstanding point of interest in the microscopy of the growth is the preponderance of the squamous-celled variety. Robb, in an analysis of 76 cases, found 72·5 per cent were squamous-celled, 21·2 per cent adenocarcinomata, 3·5 per cent papillary, 1·5 per cent columnar-celled, and 1·3 per cent transitional-celled. Bieberbach and Peters state that 60 per cent are squamous-celled, and other writers are of the same opinion. Normally the epithelium lining the perineal part of the urethra (the common site for the disease) is columnar, but Sobotta states that it is usually of a stratified columnar type and in some individuals islets of squamous epithelium can be seen. The change is probably best explained by metaplasia consequent on the malignant transformation, although the possibility of embryonal cell-nests has been suggested. In this case, although the growth occurred in the region of the fossa navicularis, where stratified squamous epithelium is usual, it assumed the form of a transitional-celled growth, a type which might reasonably be expected in the prostatic urethra.

When the growth affects the penile portion of the urethra, the symptoms are such as might be anticipated, and are well illustrated by this case. The occurrence of a bloody discharge or hæmaturia at the commencement of micturition, together with the feeling by the patient of a nodule on the under surface of the penis, should at any rate suggest to the surgeon's mind the possibility of the diagnosis of carcinoma. At a later stage the onset of urethral obstruction will cause lessening or diversion of the urinary stream, followed by complete stoppage.

In the perineal urethra the diagnosis is in most cases not made till late, and the explanation does not require exhaustive search. The association of

gonorrhœa and urethral stricture naturally focuses the attention on this condition alone, while, owing to the depth of the urethra and its inaccessibility, attention is not drawn to any nodule of tumour until it is of considerable size. When the growth has eroded the urethral wall, local extravasation and peri-urethral abscess follow very rapidly, and this, being associated with an inflammatory stricture or the passage of sounds, leads to the not unnatural adoption of the simpler diagnosis. The treatment of the abscess is soon followed by fungation of the growth and the true diagnosis then becomes apparent. The use of the urethroscope in suspected cases may be of assistance.

The statement that glandular metastases are clinically late may be due to the fact that the lymphatics of the deep urethra drain into the iliac glands, where palpation is impossible. The early death of the patients, as indicated in the literature, suggests that this deep lymphatic invasion often occurs unobserved. In disease of the anterior penile urethra the inguinal lymphatics are affected, and, in the case here recorded, were palpable on one side. This enlargement, however, may be due either wholly or partially to sepsis consequent on the malignant ulceration.

In this case the treatment adopted, for the reasons stated, was a conservative amputation. In a younger patient with a growth of the penile portion, treatment by radium should be well worth trying, for a successful result would leave the patient without deformity—a very important factor from a functional and psychological standpoint.

If radium therapy fails (and provided the patient is kept carefully under observation), amputation may then be resorted to before the condition has reached the stage of hopeless inoperability. As far as I am aware, there are no published results of radium treatment of this particular lesion. Our present knowledge of the use of radium and its application to various types of malignant epithelial growths would lead one to expect a fair measure of success, as the majority of the growths recorded are of the squamous variety.

When, however, the disease is manifested in the perineal urethra, its association with peri-urethral abscess and invasion of the surrounding tissues will in most cases prevent radical treatment. For early cases the method of penis extirpation described by Huggins and Curtis should be tried, as with radium the degree of retrogression may be difficult to observe and any recurrence easily overlooked. The very fact that an associated stricture is so prevalent makes the introduction of radium per urethram no easy matter, so that the insertion by the perineal route would be the method of choice. For advanced growths, and those opened under the misdiagnosis of simple peri-urethral abscess, radium seems to be the only method applicable.

A consideration of the results as published in the literature indicates a gloomy prognosis. The majority of patients succumb to the disease in from six months to two years. Kretschner, however, reports one case alive and well after two years. In the case reported here the time interval since operation is too short for a discussion of results. Recurrence takes place locally, with or without spread in the inguinal and iliac glands. The poor outlook must be ascribed to the usual position of the growth in the perineal urethra, which makes complete extirpation difficult, and in most cases to the

mistaken diagnosis of peri-urethral abscess. When the position of the growth is the fossa navicularis the outlook should be more hopeful.

Carcinoma of the urethra is probably not such a rare condition as the literature suggests, for it is more than probable that many cases are not recorded. Association of the condition with stricture and peri-urethral abscess should be borne in mind, as it is obviously essential to make an early diagnosis. The high proportion of squamous-celled growths in the urethra and the recent improvement in the treatment by modern radium therapy of this type of neoplastic cell in the genito-urinary system make such a method of treatment well worth a trial in an otherwise almost hopeless condition.

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A CASE OF
DIFFUSE POLYPOSIS OF THE STOMACH

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POLYPOSIS of the stomach, especially in its *diffuse* form, is a condition of such interest and rarity as to justify the addition of yet another case to the list of those on record.

The disease is characterized by the presence of numerous polypi distributed over the mucosa of the stomach ; the polypi are of various sizes and may be sessile or pedunculated. Balfour regards it as the most uncommon of all benign tumours of the stomach and states that it was found once only in 8000 operations for gastric lesions at the Mayo Clinic. Brunn and Pearl were able to collect 84 cases from the literature of the period 1820–1925, including 5 cases of their own ; their review of the subject is by far the most comprehensive of any to be found among the more recent publications. Menetrier, in 1888, was the first to give a detailed description of the disease. He recognized two forms : in the one the polypi are aggregated into a localized plaque ('polyadenomes en nappe') ; in the other they are diffusely scattered over a considerable area of the mucosa ('polyadenomes polypeaux').

The following case is an unusually good example of the diffuse form of the disease in a simple 'hour-glass' stomach.

HISTORY.—A married woman of 57, engaged in housework, who had spent all her life in England, was admitted to hospital with the following history. For fourteen years she had suffered from *indigestion* characterized by attacks of severe epigastric discomfort, or actual pain of a 'burning' nature, always made worse by the ingestion of food. In the intervals between the attacks she had been quite free from discomfort, often for many weeks at a time. For six months before admission the attacks had become more frequent and severe ; in addition *vomiting* and *diarrhœa* had supervened with increasing frequency. For many weeks these latter symptoms had been of daily occurrence ; every meal had been vomited ; the vomitus was light in colour, small in quantity, and odourless ; it contained no blood, but a great deal of 'froth' and sometimes 'slime'. The motions had been frequent, loose, and very dark in colour. She had *lost weight steadily* for the last two or three months. *Appetite* had always been good ; at times she had felt hungry, but had feared to eat because of the vomiting which would invariably follow. She had always been very moderate in the use of alcohol. She had never miscarried and had given birth to a healthy child.

ON EXAMINATION.—The patient was of the thin muscular type with a sallow complexion. The abdomen, chest, and nervous system showed no abnormality. The blood-pressure was 110 systolic and 80 diastolic ; the radial and temporal arteries were soft and elastic. Digital examination of the rectum revealed nothing abnormal. Blood examination showed : red

cells, 5,500,000 ; white cells, 13,000 (polymorphs, 64 per cent) ; hæmoglobin, 100 per cent ; colour index, 0·9 ; blood group, III. No abnormal cells were seen. The Wassermann reaction (blood) was negative. X-ray examination after a barium meal showed a well-marked 'hour-glass' deformity of the stomach ; both loculi were large with regular contours, and the channel between them was very narrow. A barium enema revealed no abnormality in the cæcum, colon, or rectum.

A diagnosis of simple 'hour-glass' stomach was made and operation advised. Unfortunately a test-meal was not given at this stage of the investigation.

OPERATION.—Operation under general anæsthesia disclosed a well-developed 'hour-glass' stomach ; the constriction lay well above the middle of the organ, and, though narrow, it was not particularly indurated ; both loculi were large, especially the distal one, which gave to the examining fingers the impression of considerable hypertrophy. The stomach was low in position and free from adhesion to neighbouring structures ; its peritoneal coat presented a normal appearance ; there was no localized induration anywhere apart from the slight thickening caused by the constriction. There was no enlargement of glands in the omenta, at the cœliac axis, or in the hilum of the liver. Examination of the rest of the abdominal contents showed no abnormality other than a general visceroptosis. Owing to the low position of the stomach and its freedom from adhesions, a partial gastrectomy by the Balfour method (jejunum in front of colon with efferent loop from greater curve) was carried out without difficulty, the section through the proximal loculus being made 1½ in. above the constriction. When the stomach was divided it was noticed that its mucosa was thicker than usual and studded all over with minute polypi, sessile in form. The mucosæ of the duodenum and jejunum were normal. Approximately two-thirds of the stomach was resected.

The patient made a satisfactory recovery from the operation and left the hospital four weeks later, stating that she felt better than she had for months previously. Her colour and general appearance had improved ; she was able to take her meals without discomfort ; the vomiting had ceased ; and the bowels were acting normally.

A test-meal was given during the fourth week of convalescence and showed a total achlorhydria. A second blood examination carried out at the same time showed : red cells, 4,500,000 ; white cells, 4,700 ; hæmoglobin, 78 per cent ; colour index, 0·85.

Before she left hospital the rectum and colon were examined with the sigmoidoscope and nothing abnormal was detected.

When seen eight months after the operation she looked well ; she had gained in weight ; she was taking an ordinary diet and enjoying it ; and, with the exception of some slight frequency of micturition, she had no symptoms whatever.

DESCRIPTION OF SPECIMEN.—The distal loculus is large ; it measures 5 in. along the lesser curve. The aperture of the constriction is narrow and only just admits the forefinger ; its diameter is ¾ in. The duodenal mucosa and musculature are normal right up to the pylorus, where an abrupt change
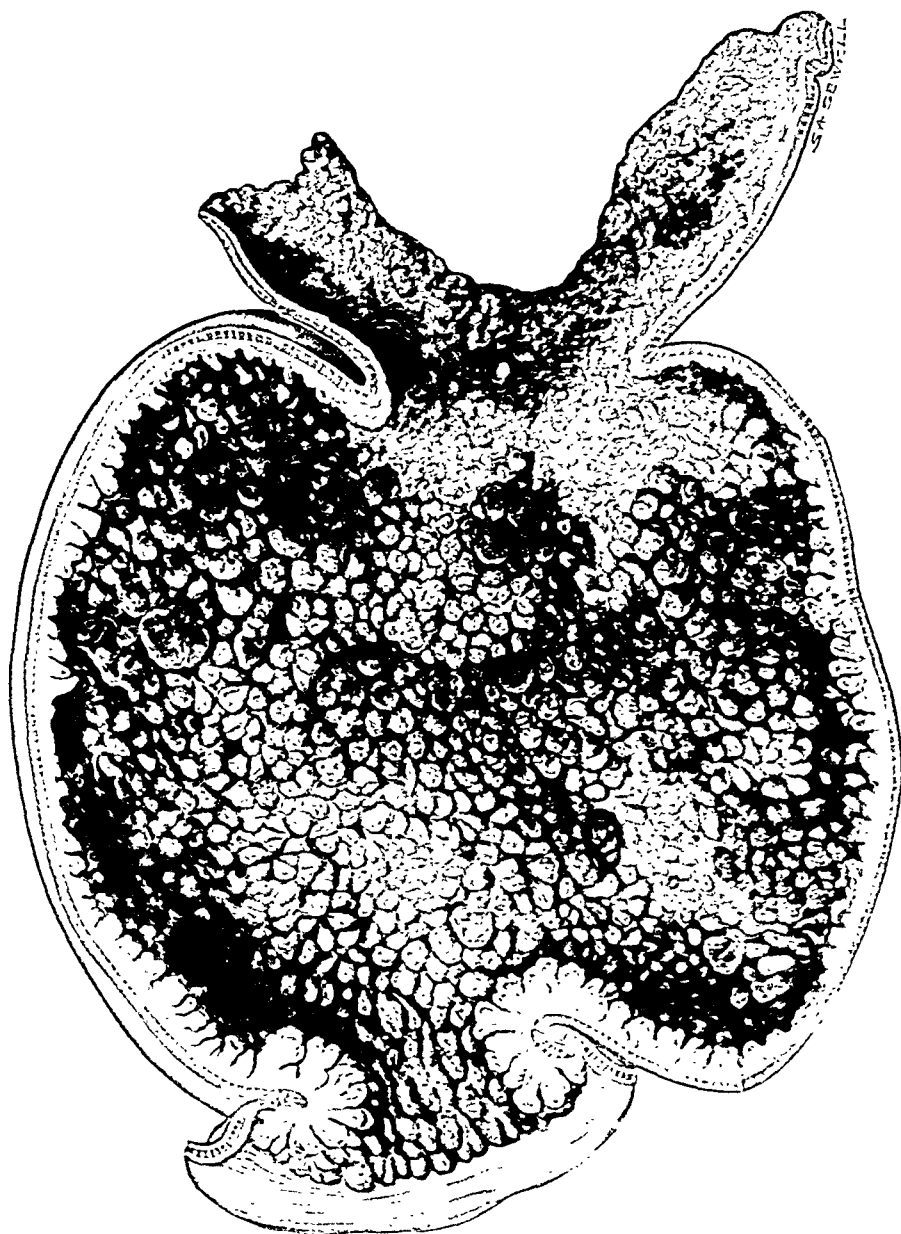
FIG. 408. Showing the interior of the specimen exposed by an incision along the greater curve. On the left is seen the normal mucosa of the duodenum, contrasting sharply with that of the pylorus, which is polypous and prolapsed. On the right is seen the 'hour-glass' constriction. ($\times \frac{2}{3}$.)

is evident, for through the canal numerous dark-coloured polypoid masses project, resembling a number of prolapsed and œdematous piles. The polypi have definitely obstructed the lumen of the pyloric canal, which just allows the passage of a pair of Spencer Wells artery forceps. The peritoneal covering on both anterior and posterior surfaces of the specimen is normal. A few small and apparently normal glands are present in the gastro-hepatic and gastrocolic omenta. The interior of the specimen, when exposed by an incision along the greater curve, exhibits a diffuse pathological change which differs somewhat in the two loculi (*Fig.* 408). The distal loculus is covered with closely-packed polypoid projections of the mucous membrane, mostly about ⅓ in. in width and height ; most of the projections are rounded, but in some areas they are flattened or elongated by pressure ; some are pedunculated and others are sessile ; a few attain a diameter of ½ in. ; there are groups of polypi of a deep red colour from congestion or extravasation of blood, giving the general appearance of a patchy·inflammation. With a reading lens many smaller polypi of various sizes are seen lying between the larger ones ; as many as forty polypi to the square inch may be counted in several areas. In the proximal loculus, on the line of constriction, and over a small area adjacent to the constriction on its pyloric side, the mucous membrane is slightly thickened, having the 'mammillated' appearance of chronic gastritis ; here the projections are small and mostly sessile in form. The mucosa of both loculi is covered with a thin layer of mucus. There is no evidence of ulceration anywhere. The muscular coat of the stomach is slightly hypertrophied, but nowhere is any localized induration felt, except at the constriction, which is just palpable as an even and slightly thickened ring.

MICROSCOPIC EXAMINATION.—

The Distal Loculus.—Two sections were examined ; one through the pyloric sphincter and adjoining portion of the duodenum (*Fig.* 409) ; another from the region of the greater curve at the middle of the loculus. The mucosa is thickened and polypous ; the polyps, in transverse and longitudinal section, are seen to be composed chiefly of mucosal crypts with abundant stroma ; occasional crypts are distended to form small cysts. The epithelial cells are typical. The deeper part of the mucosa is composed of typical fundal or pyloric glands, which rarely pass up a short distance into the polyps ; in the fundal glands parietal cells are numerous. There are hæmorrhages in the summits of many of the polyps ; their stroma is œdematous and its capillaries are distended. The stroma is densely infiltrated with plasma cells, eosinophil leucocytes, and lymphoid cells, and in places with neutrophil leucocytes. There are also numerous large round cells whose cytoplasm is occupied by hyaline eosinophilous bodies (Russell bodies). In one polyp these cells are very numerous, filling the greater part of the stroma. The infiltration is somewhat less in the deeper parts of the mucosa between and deep to the glands ; here there are a few lymphoid nodules. There is a moderate degree of fibrosis, with few spindle cells, throughout the mucosa. The muscularis mucosæ is normal except for slight inflammatory infiltration similar to that in the mucosa. In the submucosa and muscularis externa there is a similar but very slight and focal infiltration. A few mast cells are present in the muscularis externa.

FIG. 409.—Section through the pyloric sphincter, showing polypi in transverse and longitudinal section.   In the lower part of the drawing the normal mucosa of the duodenum is seen.   (× 3.)
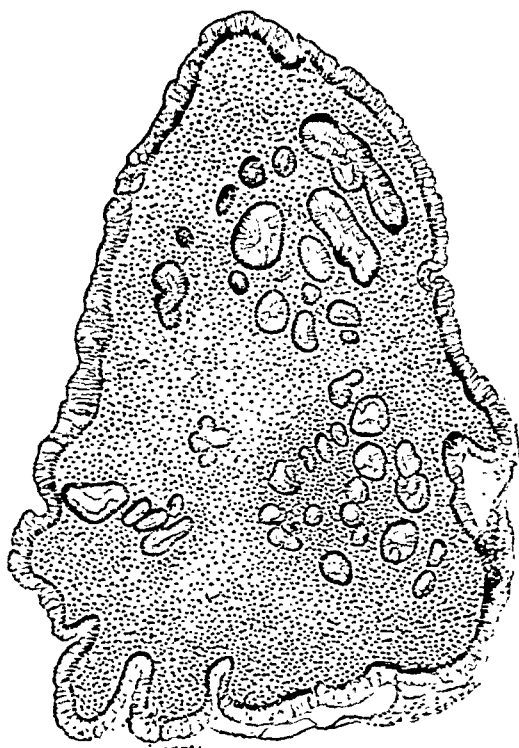
FIG. 410.—A polyp in transverse section   (· 60.)

The microscopic appearances suggest that chronic inflammation has produced polypous thickening of the mucosa. The mucosa of the duodenum is not thickened ; it is densely infiltrated with plasma cells and eosinophil leucocytes. *Fig.* 410 shows a polyp in transverse section.

The Proximal Loculus.—One section was examined from the region of the greater curve. The mucosa is not polypous and is thinner than that of the distal loculus. Its stroma is œdematous, congested, and infiltrated with plasma cells, lymphoid cells, and eosinophil leucocytes ; there are a few cells containing Russell bodies and very few neutrophil leucocytes. These features are much less conspicuous than in the distal loculus. The other coats are normal.

The Constriction.—A section was made from the lesser curve in a longitudinal direction, cutting the constriction transversely. At the small area of constriction there is great fibrosis of the lesser omentum and of the muscularis externa, though everywhere in this scar in the muscularis externa there are at least small bundles of muscle fibres. The muscularis externa is fused with the muscularis mucosæ, the fibres of the latter being separated by increased fibrous tissue. Spindle cells in the scar tissue are small and not numerous and there are very few round cells. The mucosa is thin but complete over the scar. The thickened polypous mucosa of the distal loculus on the one side of the scar contrasts conspicuously with the thinner mucosa of the proximal loculus on the other side.

Lymphatic Gland.—Only one lymphatic gland (0·5 × 0·3 cm.) was attached to the specimen. Its sinuses are broad, with many endothelial cells. The peri-adenoid connective tissue is slightly infiltrated with plasma cells.

There was no evidence of malignant change in any of the sections examined.

Etiology.—The etiology of the disease is obscure. There is some evidence that a *congenital* factor may be present, for it has been found in twins, in brothers and sisters, and in mother and son ; in this connection it is of interest to note that there is a distinct familial tendency in the analogous condition of polyposis of the small intestine and colon. Syphilis, tuberculosis, alcohol, and arteriosclerosis have all been blamed, but there is no real evidence that they are important etiological factors.

According to Brunn and Pearl the disease occurs most frequently in the sixth and seventh decades of life ; the youngest case reported was in a girl of 20 ; males are more often affected than females. The condition is sometimes associated with generalized polyposis of the intestinal tract ; this was so in 7 per cent of Brunn and Pearl's cases.

The true *nature of the morbid process* is still a matter of uncertainty. Evidence of this is to be seen in the variety of names given to the disease : gastritis polyposa, adenomatosis gastrica, papillomatosis gastrica, polyadenoma gastrica, diffuse gastric polyposis, and adenopapillomatosis gastrica. The tumours are generally referred to as ' adenomata ' ; they are covered by a single layer of columnar or cuboidal cells arranged in an orderly manner and limited by the muscularis mucosæ. In many of the cases recorded there was evidence of *chronic inflammation* of the gastric mucosa. Considerable stress is laid by

some writers on the presence of polynuclears and lymphocytes in the stroma, while others specifically record their absence. Menetrier insists that the polyposis is secondary to chronic gastritis. while others regard the inflammatory changes as secondary—a view which is supported by the fact that polyp formation is rare in cases of chronic gastritis. Mills considers that the tumours should be designated 'papillomata' rather than 'adenomata', as no capsule is present ; he further suggests that they may be of the nature of infected warts similar to those found on the skin. Brunn and Pearl found evidence of chronic inflammation in only 32 per cent of their cases ; they consider that the term 'adenopapillomatosis' most accurately describes the pathological picture, and that the disease may originate in two ways : (1) As a congenital hypertrophy of the epithelium ; (2) As a hyperplasia acquired through chronic inflammation. One writer claims to have discovered all stages from hypertrophic gastritis to adenomatous formation and even to carcinomatous change (Konjetzny).

That *chronic irritation* can produce polypoid growths in the stomach seems proved by the experiments of Fibigen and Wassink, who produced diffuse polyposis in the stomachs of rats by feeding them on cockroaches infected with the larvæ of certain nematodes ; they were able to follow the lesions through from the initial stages of simple epithelial hyperplasia to malignant degeneration. Papillary adenomata have also been produced experimentally by the injection of coal-tar into the submucosa of rabbit's stomachs (Ishibashi and Ohtani).

It is of interest to note that the specimen described by the present writer is classified in the Museum of the Royal College of Surgeons of England under the heading of 'chronic hypertrophic gastritis'. The sequence of events in this case was probably as follows : the patient had a gastric ulcer which healed and produced a bilocular stomach ; in association with this an unusual degree of chronic gastritis developed in the distal loculus ; this led to thickening of the mucosa and the formation of inflammatory polyps ; these in their turn caused progressive obstruction of the pylorus with consequent stasis and further gastritis ; and in this way a vicious circle was established. It is suggested that these polypi should be regarded as blastomatoid in origin rather than as true tumour formations.

Association with Carcinoma.—It is generally agreed that polyposis of the stomach is not commonly associated with carcinoma. The two diseases were present together in only 12 per cent of Brunn and Pearl's cases. Of 263 cases of carcinoma of the stomach examined by Stewart, only 13 were associated with polypi (4·9 per cent). This is in marked contrast with the case of the colon and rectum. where the two conditions co-exist much more frequently ; thus of 33 cases of carcinoma of the colon and rectum examined by Dukes. as many as 25 showed associated polyposis (75 per cent). Stewart concludes that : " Simple adenomatous polypi of the stomach. whether single or multiple. must therefore be accepted as a definitely precancerous lesion. but the available evidence goes to show that the association is much less intimate than in the case of the large intestine."

The Diagnosis of Gastric Polyposis.—In the majority of the cases reported the specimens have been obtained from the post-mortem room. the

disease having been unrecognized during life. In recent years operation has led to the discovery of an increasing number; in only a few instances has the diagnosis been made before operation. There are no characteristic symptoms whatever. In a few cases the diagnosis has been made on X-ray examination (e.g., Carman in 1917 and Balfour in 1919); the characteristic picture is one of irregular defects in the contour of the stomach due to indentation by the polyps; this appearance in a case of chronic dyspepsia with achlorhydria and the vomiting of blood-stained mucus is suggestive of polyposis. It is to radiography, therefore, that we must look in the future for precision in the diagnosis of this uncommon disease. One case is reported in which the condition was recognized by the gastroscope (Schindler, 1922); in another (Wegele, 1909) the finding of a polyp on the tube used for gastric lavage led to the diagnosis. In those cases in which test-meals have been carried out *achlorhydria* was a constant finding.

SUMMARY.

1. A case of diffuse polyposis in a simple 'hour-glass' stomach is described.

2. The main symptoms were those of a chronic dyspepsia on which had supervened the more acute symptoms of persistent vomiting and diarrhœa.

3. The condition was not recognized until the specimen had been removed by operation.

4. The microscopic appearances suggest that chronic inflammation had produced polypous thickening of the gastric mucosa.

5. There was no evidence of malignancy.

6. The disease is uncommon; only 84 cases have hitherto been recorded.

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THE TREATMENT OF ACUTE EMPYEMA IN INFANCY AND CHILDHOOD.

WITH A REPORT OF 75 CASES TREATED BY CLOSED DRAINAGE.

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EMPYEMA THORACIS is a common disease of childhood. Three per cent of admissions to the surgical wards of our Children's Hospital are for this condition. With us it is more common than acute appendicitis. The high mortality with which it is attended, especially in the first two years of life. may be taken as sufficient proof that treatment has been unsatisfactory. The following table gives the death-rate from acute empyema in children under two years of age in some representative American hospitals These statistics warrant continued search for new or improved methods of treatment in this disease, which has taken such a marked toll of life in the period of infancy.

HOSPITAL		PERIOD	MORTALITY
			Per cent
Baby's Hospital, New York	..	1903–13	37·8
Bellevue Hospital	..	1920–4	34·8
Children's Hospital. Boston	..	Prior to 1924	35·0
Johns Hopkins Hospital	..	1889–1927	29·6
Johns Hopkins Hospital	..	1927–9	22·2

More than forty years ago Beulau inserted a catheter through a trocar into the thorax. and applied negative pressure drainage. While the occasional surgeon has come forward from time to time to advocate the closed method of treatment in empyema. it has not had very wide application. A considerable number of operators have tried it in a few cases, only to abandon it when technical difficulties in its application were met with. One of the main difficulties was leakage around the catheter. which occurred in a few days or at best a week. Trouble was also experienced on account of the opening in the catheter becoming plugged with lumps of fibrin. The apparatus often leaked and otherwise went out of order. In a considerable number of cases rib resection was performed as a way out of the difficulty.

The author has used the method of applying closed drainage here described in seventy-five consecutive cases and has found the results obtained to be so much better than those experienced with the open method that the latter has been entirely abandoned. Nothing original is claimed either for method or for apparatus. We have worked out a mode of application that

has proved satisfactory with us, and I feel entirely confident that it will prove to be just as satisfactory in the hands of others, if sufficient attention is paid to the details of its use. It has already been described briefly in a paper read by me at the meeting of the British Medical Association in Winnipeg in 1930 which was later published in the *British Medical Journal*. It is described here in more detail, with correction of some minor errors in the illustration of the apparatus which were unfortunately overlooked at that time. Thirty-three additional cases are added, making seventy-six cases in all treated by the method.

Regardless of what form of treatment is used in empyema there are certain principles which, I think all surgeons will agree, must be kept in mind. First, the operation to provide drainage must be delayed long enough for the lung to become firmly adherent, in order that lung collapse may be prevented when the thorax is opened. Secondly, the more acute stage of

FIG. 411.—Showing a case of empyema with lung collapse as a result of air being allowed to enter the chest during paracentesis.

the illness should be over, with improvement in the general condition and the pneumonic process resolved before the child is subjected to operation.

How, then, is one to know when adhesions between lung and chest wall are firm enough to prevent lung collapse, with enlargement of the cavity and spread of infection, when air is allowed to enter the thorax? We know that in cases of pneumonia due to the pneumococcus involvement of the pleura occurs late. Usually the pneumonia will have resolved before the empyema is discovered. Adhesions between the lung and chest wall form quickly and firmly. On the other hand, if the invading organism is the streptococcus, involvement of the pleura may occur early, perhaps on the third or fourth day following the onset of the acute illness, when the pneumonic process is

at its height and when the lung adhesions are absent or very weak. Open thoracotomy at this stage with massive collapse of the lung must be looked upon as a catastrophe. We have the following rule at the Children's Hospital as a practical working basis : When the organism present is the pneumo-coccus, *thick* pus must be present in the pleural cavity for at least a week before the empyema is drained ; when we are dealing with the streptococcus, *thick* pus must be present for, at least ten days before drainage is undertaken. While we are waiting the child is kept comfortable by removal of the pus by aspiration as often as we think necessary, depending upon the rapidity with which it re-accumulates.

A word about aspiration of the chest may not be out of place here. A considerable number of our cases admitted to hospital have had the chest aspirated either for diagnosis or treatment. In a number of these we have found air present in the empyema cavity, sometimes in small amounts, occasionally in large quantities with marked collapse of the lung (*Fig.* 411). We are firmly of the opinion that the introduction of air into the cavity in
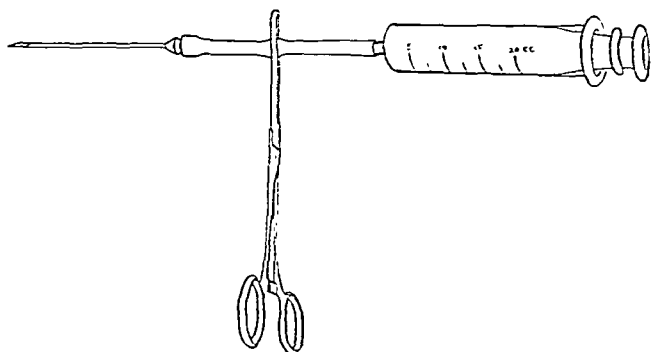
Fig. 412.—Showing method used in aspirating chest.

this manner in the early stages of the disease causes spread of infection and forms an additional menace to the life of the patient. It is our experience that these patients run a stormier course than those in which the accident has not been allowed to occur. To aspirate the chest either for purposes of diagnosis or treatment we use the simple apparatus illustrated in *Fig.* 412. It consists of a good-sized needle with a piece of rubber tubing about 5 in. long pushed on over the end. An artery forceps clamped on this tubing prevents the entrance of air when the needle is being introduced into the chest and when the syringe is detached for emptying. By using a large syringe any amount of pus may be withdrawn quite conveniently. If the syringe is attached directly to the needle. leakage of air frequently occurs at the point of connection : the same applies when the tubing is attached to an adapter which fits into the needle. The entrance of very large amounts of air is. however. usually due to the end of the needle being left unprotected when the syringe is removed for emptying.

Apparatus.—The apparatus which we use for the application of suction and irrigation is very simple. It consists of three ordinary Winchester

bottles, with tight-fitting rubber stoppers pierced with two holes, some glass tubing, a container for Dakin's solution, and some rubber tubing of suitable size, all of which may be obtained at any chemist at a small cost (*Fig.* 413). The A bottle is used simply as a trap to prevent the washings from the empyema cavity from becoming mixed with the water from the siphon. It is essential that the corks fit tightly in the bottles and that the glass tubing fit tightly in the corks, otherwise difficulty will be experienced in maintaining the suction on account of leakage of air. If leakage about the corks does occur, it can usually be stopped in the following manner : Place a cuff of adhesive plaster about the neck of the bottle so that the top of the adhesive
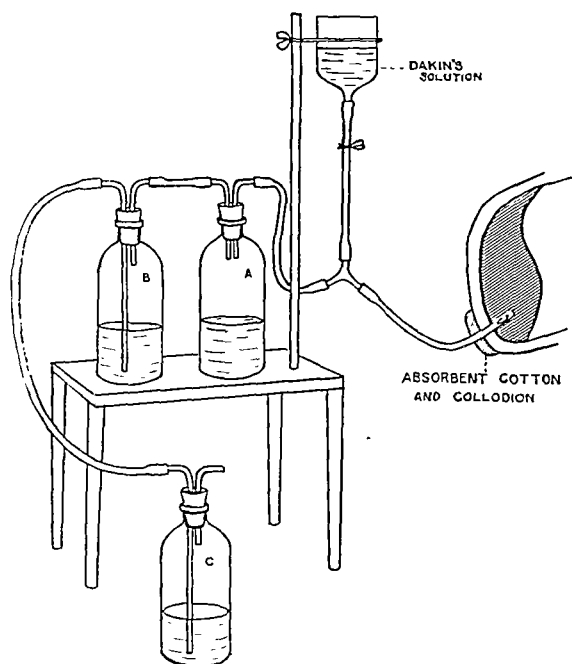
FIG. 413.—Closed drainage in empyema. To irrigate the cavity the rubber tube connecting the Y glass connection with bottle A is clamped, after which the clamp is removed from the tube connecting the Y with the container for Dakin's solution ; when the abscess cavity is filled the solution is clamped off and the clamp betweed the Y connection and bottle A removed, allowing the solution to drain off by suction into bottle A. To remove the solution accumulated in bottle A, clamp both rubber tubes leading from the glass connections in the cork ; the cork may then be withdrawn and the bottle emptied without disturbing the siphon. Bottles B and C are interchangeable. When bottle B is nearly empty place a clamp on the rubber tubing between bottles A and B and another clamp in the tube between bottles B and C ; the bottles are then changed, placing the empty one on the floor and the full one on the table.

extends about $\frac{3}{4}$ in. above the top of the cork. Now melt some grocer's paraffin to which a small quantity of vaseline has been added, and when it has become liquid pour it into the cup formed by the adhesive plaster. This will effectively seal all openings through which air might enter. This procedure is not recommended as a routine, for it has to be repeated if the cork is removed from the bottle or if the glass tubing is disturbed to any great extent.

Technique of Operation.—The operation is performed under local anæsthesia and can be carried out quite readily in bed if the patient is too ill to be moved. In the case of infants and children a catheter size No. 16 or No. 18 (French) is used. This catheter fits so snugly into the cannula that when it is lubricated with glycerin no air can enter around it. An artery forceps clamped on the catheter near the end prevents the entrance of air through its lumen. The trocar with cannula is introduced between the ribs at

the most convenient point. The trocar is then withdrawn and immediately the index finger is placed over the end of the cannula to exclude air. The operator now takes the catheter in his right hand, removes his finger from the end of the cannula, and at once inserts it. In this way the catheter is introduced into the pleural cavity without the admission of air, excepting the small amount that might enter upon removal of the trocar and insertion of the catheter.

Even though one feels that the lung is attached to the chest wall by adhesions sufficiently firm to prevent its collapse if air was allowed to enter, every possible precaution is taken to avoid this undesired complication, for we look upon lung collapse in empyema as nothing short of a calamity. The catheter, which has a lateral eye $\frac{1}{2}$ in. above the terminal one, is allowed to project into the cavity $1\frac{1}{4}$ in. In order that one may know its position in the chest a mark is necessary. If, before beginning the operation, the operator places the catheter beside the cannula allowing it to project the required $1\frac{1}{4}$ in. beyond its end, and with the scissors removes a small chip from its wall opposite the distal end, he will have a landmark that is quite satisfactory. With the catheter now in place the cannula is removed.

Before removing the clamp on the end of the catheter to take off the cannula a clamp is placed on the proximal side of the cannula for obvious reasons. Then comes the very important step of sealing the opening about the catheter. This is accomplished by applying successive layers of absorbent cotton to which collodion is applied with a brush until a collar, 5 in. in diameter and $\frac{1}{2}$ in. thick, is built up about the tube. In order that this dressing may adhere firmly to the skin and adjacent catheter these parts must be thoroughly clean and dry. This is brought about by washing them carefully with ether. To begin the application of the dressing a layer of collodion is applied to the skin with the brush. A thin layer of cotton is then laid on this base and smoothed out with the brush which has been dipped in the collodion. This ensures good cohesion between the dressing and the skin. By the application of successive layers of cotton in a similar manner the dressing is built up to the required thickness. We have found that the dressing will remain tight longer if the catheter is held immovable in the chest wall. This is accomplished in the following manner: When the dressing is about half applied a small round needle threaded with a double strand of silk is passed through the wall of the catheter (without entering its lumen) flush with the surface of the dressing. This double thread is then cut, leaving the ends about 2 in. in length. Another thread is then passed through the opposite wall of the catheter in a similar manner. These threads are embedded in the dressing as it is completed and effectively prevent movement.

To ensure that the dressing will not be disturbed in moving the child back to bed, it is fanned for twenty minutes before the patient is moved from the operating-table. The child is then returned to the ward with the clamp still on the end of the catheter. To allow the dressing to set more firmly this clamp is left in position for three hours longer, when the catheter is connected up with the apparatus and the clamp removed, and the pus allowed to drain off. If the effusion is large and the surgeon wishes to decompress

the lung slowly, this can be done by removing and applying the clamp at intervals as desired.

Irrigation.—Irrigation of the cavity is begun the following morning—that is, twenty-four hours after the catheter has been inserted. These irrigations are carried out every two hours during the day and three times at night. The top of the fluid in the container for Dakin's solution is never allowed to be more than 18 in. above the opening in the chest, as too much pressure tends to force fluid out around the tube under the dressing, thus loosening it from the skin. To irrigate, a clamp is placed on the rubber tube leading to the A bottle, then the clamp is removed from the tube leading from the Dakin's flask and the solution allowed to run into the chest until it causes pain or stops from the equalization of pressure. This clamp is then replaced and the one on the tube leading to the suction apparatus removed, when the contents of the cavity drain off into the bottle marked A.

If, as sometimes happens, the eye of the catheter becomes plugged with fibrin or thick pus, it can often be freed by stripping the tube in such a way as to force a small amount of solution into the cavity, or, if this fails, by working in opposite fashion, increasing the negative pressure to draw some out. Should this prove unsuccessful, disconnect the catheter from the drainage apparatus, then with a syringe containing Dakin's solution or saline, inject and withdraw small amounts of fluid. This manœuvre is nearly always effective. In only two cases was it necessary to remove the catheter and perform the operation over again, and in both of these cases I had failed to provide the catheter with the lateral eye.

In about 8 per cent of our cases blood appeared in the washings from the cavity. If the return is only slightly tinged, it will probably be sufficient to reduce the amount of negative pressure. This is accomplished by raising bottle C a foot or more from the floor. If, however, blood appears in any considerable quantity, it is advisable to discontinue the irrigation for twenty-four hours. In our experience hæmorrhage has never been a serious complication and has always been readily controlled by the above means.

Occasionally we meet with a case in which the introduction of the solution into the pleural cavity sets up troublesome spasms of coughing. These occur usually at the beginning of treatment and disappear after three or four days. The coughing as a rule begins when the cavity is about filled with solution. By allowing the fluid to run in very slowly and stopping before the danger-point is reached these patients can be carried along until this troublesome symptom disappears.

If a bronchial fistula is present, treatment will have to be adapted to meet that complication. In some of these cases, while the irrigating fluid will enter the bronchus, air from the bronchus will not enter the cavity, so that though irrigation cannot be carried out, yet drainage can be continued with negative pressure. In these cases the irrigations are discontinued until the fistula heals. Sometimes one finds that a certain amount of solution can be allowed to run into the cavity without causing coughing—that is to say, the cough is not brought on until the fluid reaches the level of the opening in the bronchus. By changing the position of these patients one sometimes finds a posture in which a considerable amount of fluid will run into the

cavity without causing coughing. If this can be done, irrigation of the cavity in this modified way is carried out until the fistula heals. If air passes from the bronchus to the cavity, of course negative pressure cannot be maintained. Then a clamp is placed on the catheter, which is removed for a short time every three hours to allow the escape of air or discharge that might be accumulating in the cavity under pressure. When the fistula heals treatment is continued in the regular way.

A question I am often asked, and one to which it is difficult to give a fixed answer, is—When shall the irrigations be discontinued and the catheter removed from the chest ? In the average case the temperature drops to normal in two or three days, the cavity gradually becomes smaller, and the amount of pus in the washings rapidly diminishes. The size of the cavity and the amount of pus in the returns are the principal factors which determine the time at which drainage may safely be discontinued. Usually when the cavity holds not more than 15 c.c. of fluid, and when the returns have been free from macroscopic pus for five days, we feel that drainage can be safely discontinued. While this rule will be found satisfactory in most cases, yet something must be left to the judgement of the surgeon in individual cases.

A case comes to mind in which empyema followed spontaneous pneumo-thorax. The cavity was very large as there was complete collapse of one lung. The patient, a boy, had a bronchial fistula. Following the healing of the fistula, which took place in seven days, irrigations with the continuous application of negative pressure were carried out in the routine way. The lung expanded until the cavity held 8 oz. At this point, however, progress seemed to be arrested in so far as reduction in the size of the cavity was concerned, though the washings were returning free from pus. Notwith-standing the large size of the cavity remaining, the catheter was removed from the chest and a dry dressing applied. This boy made an uneventful recovery, the lung came out to the chest wall later, and he has remained well during the four years which have passed since.

Our routine treatment of the wound after removal of the catheter is to wash the area with alcohol on a sterile sponge and apply a dry dressing.

Results.—Seventy-five consecutive cases of acute empyema in infancy and childhood were treated by the method here described. In 46 cases the organism was the pneumococcus, in 9 the streptococcus, in 6 the staphylo-coccus, in 1 case a mixed infection of pneumococcus and streptococcus, and in 1 a mixed infection of pneumococcus and staphylococcus. Six cases were reported 'no growth', and in 6 no report could be found. The following group shows the number of cases in each year of life :—

Year	Number	Year	Number
1st	7	7th	6
2nd	14	8th	4
3rd	10	9th	4
4th	6	10th	7
5th	5	11th	1
6th	11		

The youngest child was 4 months and the eldest 11 years. There were two deaths, both in children under 2 years of age, making a mortality for the

series of 2·66 per cent, and a mortality for those under 2 years of age of 9·52 per cent.

One death already reported occurred in a child 14 months old who developed empyema following bronchopneumonia. Vomiting was an outstanding symptom and persisted from the time of admission until death. Necropsy revealed that this child had developed pneumonia of the other lung and that acute pericarditis was also present.

The second occurred in a child 19 months old who was admitted with empyema which arose as a complication of an acute illness which had been present for three weeks and had been diagnosed as influenza. The temperature on admission was 103·4°, and two days later rose to 105°. Bilateral otitis media was present, the left ear discharging and the right drum bulging. A needle was introduced into the chest and 150 c.c. of greenish yellow pus were aspirated which on culture showed streptococcus. The child's condition was very grave and he was vomiting almost continuously. The abdomen was distended. Three days after admission pneumothorax was diagnosed, and on aspirating the chest another 150 c.c. of pus were withdrawn mixed with considerable quantities of air. The air in the chest rapidly re-accumulated, thus embarrassing respiration. As it was found impractical to keep the air removed by repeated aspirations a catheter was introduced in the routine way. Negative pressure could not be maintained, as the air entered the cavity freely through the bronchial fistula. No attempt at irrigation was made. Air and pus escaped through the catheter freely. The patient's condition grew steadily worse—temperature 105°, pulse 160. Vomiting continued until death, which occurred on the tenth day after admission. The parents refused permission for autopsy.

Both children who died presented pictures which were strikingly similar to that seen in fatal cases of infantile cholera. They had the same dehydrated appearance with loose skin and sunken eyes. Vomiting was an outstanding symptom in both, and persisted until death.

There were 7 cases of bronchial fistula. In 4 of these the fistulæ were present before the catheter was introduced into the chest. They all healed in from three to seven days.

There were 3 cases of pocketing. Two of these were discovered while the child was still in hospital, and have already been reported. The third occurred in a child of 9, who returned six months after operation with a pocket situated some three inches above and medial to the original drainage site. Closed drainage in the routine way was instituted, and uneventful recovery followed.

Cellulitis or abscess of the chest wall occurred in 7 cases. In no case did this complication occur as a result of the introduction of the catheter, all having followed aspiration. In 3 cases this condition was present when the patients were admitted to hospital. Recently we have been injecting mercurochrome into the track of the needle with the hope of reducing the incidence of this troublesome complication.

The average number of days of drainage for the seventy-five cases was 21·8. With the exception of the cases of pocketing there were no cases requiring secondary operations, nor were there any instances of chronic empyema.

COMMENT.

The treatment of empyema by the method here described requires more attention to detail on the part of the surgeon than does treatment of the condition by rib resection; but if he is willing to give that extra care, he will be well repaid in the saving of life, the shortening of the period of illness, the leaving of a more normal and better functioning chest wall, and in the comfort he will bring to his patient in the avoidance of painful dressings. This latter advantage alone would, to my mind, be sufficient reason to use the closed method in preference to open drainage. The shortening of

FIG. 414.—Showing a case of encysted empyema.

the time of drainage has considerable economic value. If we take six weeks as the average time of drainage in rib resection. we have by the use of the closed method cut the time in half. We have demonstrated over and over again that for practical purposes the empyema cavity can be rendered sterile by the use of Dakin's solution. It has been said that irrigations with Dakin's solution will increase the incidence of bronchial fistula. We have had no evidence to support that conclusion. In 4 out of the 7 cases which developed, the fistula was present before the catheter was introduced into the chest. For the treatment of encysted empyema (*Fig.* 414) the method is ideal. In not a single case have we found it necessary to resort to rib resection.

CONGENITAL DISLOCATION OF THE HIP IN A CASE OF MULTIPLE CONGENITAL DEFORMITIES.

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A FEMALE child (O. S.), aged 3 years and 4 months, was admitted to Pyrford Orthopædic Hospital in June, 1931, with multiple congenital deformities. She was small for her age and backward, her mental age being that of a child of about 18 months. She showed the widespread failure of normal development of voluntary muscles, with associated contractures of joints, characteristic of the condition known as ' arthrogryphosis multiplex congenita '. The muscle groups most markedly undeveloped were the quadriceps femoris, hamstrings, anterior and posterior tibials, small muscles of the feet, and the muscles of the anterior abdominal wall. There was a marked lordosis of the lumbar spine. Both hips were in a position of fixed flexion of 60°, the left hip being also in fixed adduction of 30°. The left hip was congenitally dislocated. The knees were in full extension with only a few degrees of movement, in contrast to the fixed flexion deformity which is more common in this condition. The right foot was in a position of severe talipes equinovarus ; the left showed an equally severe degree of calcaneo-valgus.

On admission the child was very ill with bronchopneumonia. She developed a streptococcal peritonitis, and died three months later. The pelvis and hip-joints were available for investigation after death. Congenital dislocation of the hip-joint in arthrogryphosis multiplex congenita is always extremely difficult, if not impossible, to reduce by manipulation, and an examination of the hip-joints was therefore made to ascertain the particular factors concerned in producing this clinical differentiation from the majority of the more ordinary type of congenital dislocation of the hip. *Fig.* 415 is a diagrammatic drawing of the left hip-joint opened and seen from above and behind.

Description of Specimen of the Left Hip-joint.—As may be seen in the X-ray of the specimen (*Fig.* 416), all the bones of the pelvis, as well as the upper end of the femur, are in a retarded state of ossification on the left side as compared with the right. The neck of the femur is short and abnormally anteverted on the shaft. The specimen does not contain sufficient of the shaft of the femur for the amount of this anteversion to be assessed accurately.

As far as can be ascertained by careful dissection, the muscles about the left hip are normal in their attachment and development and in the arrangement of their nerve-supply. The quadriceps and hamstrings, on the other hand, are poorly developed, and in the lower half of the thigh are mainly replaced by fibrous tissue and fat.

There is marked shortening of the hip flexors and adductors holding the thigh in the flexed and adducted position, and it is impossible to say how far these contractures might have been overcome by forcible stretching during life. The fixation of the hip in adduction is, however, very great, and it seems probable that the resistance of the soft parts alone would have made manipulative reduction extremely difficult.

The gluteal muscles have been divided horizontally above the greater trochanter and the capsule of the hip-joint is opened from above. It was difficult to find the cavity of the hip-joint, as the greatly thickened
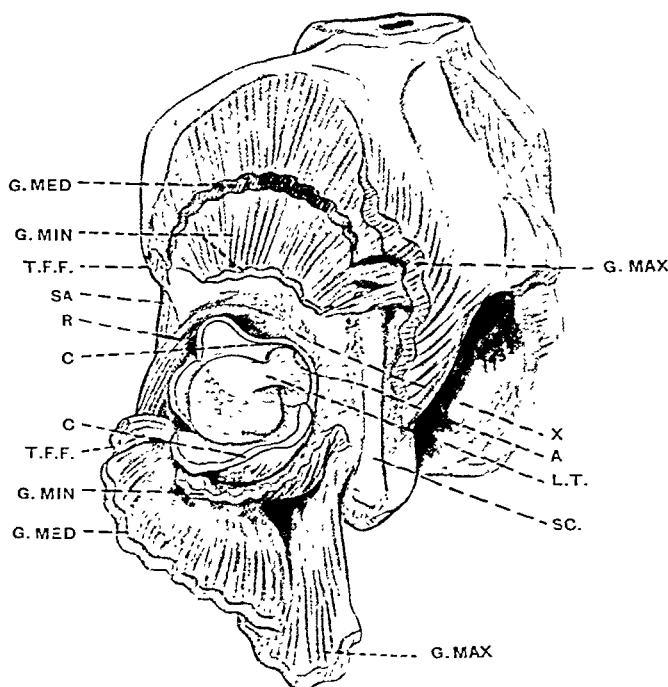
FIG. 415.—Semi-diagrammatic drawing of the hip-joint open. G. MAX, Gluteus maximus: G. MED, Gluteus medius; G. MIN, Gluteus minimus: T.F.F.. Tensor fasciæ femoris: SA, Sartorius; R, Rectus femoris (reflected head); SC., Sciatic nerve; C, Capsule of hip-joint: L.T., Ligamentum teres: A, Upper margin acetabulum: X, Area above acetabulum where outer surface of capsule is attached to cartilage of ilium and fascia over reflected hea l of rectus femoris.

capsule formed a hood over the head of the femur of almost cartilaginous consistency, and the fascia on the deep surface of the muscles was bound so intimately to this that a clear differentiation of the structures in this region was far from easy.

The dome of capsule covering the dislocated head is two and a half times the thickness of the normal capsule on the right side, and consists of dense white fibrous tissue. There is no suggestion of hour-glass contracture of the capsule producing partial separation of the joint space into two compartments. for the capsule passes directly down over the inner aspect of the
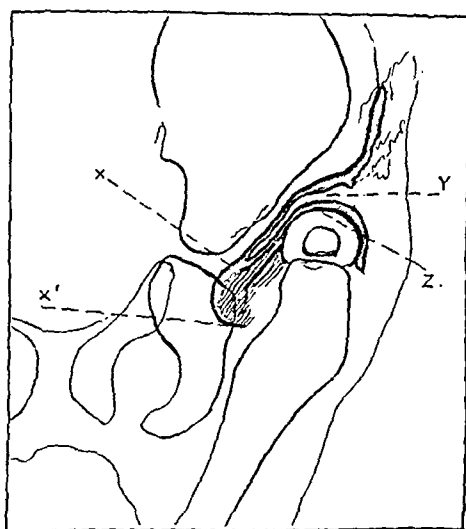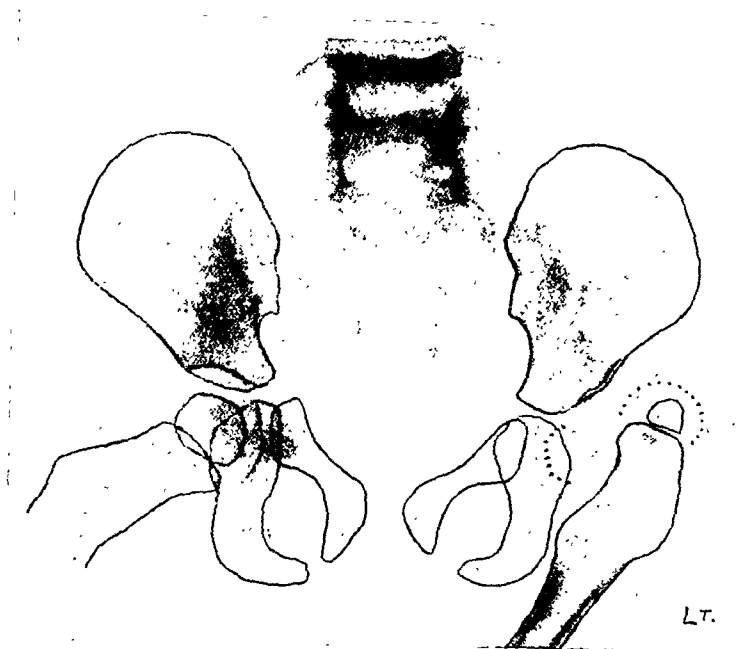
FIG. 416.—Skiagram of the pelvis, and diagram to emphasize some points in the X-ray; the left hip-joint has been opened, but the ligamentum teres is intact and the head bears the same relationship to the acetabulum as durirg life. The air in the joint outlines the cartilage of the head (Z), and it will be seen that the size of this is greater than that of the acetabulum (X—X¹), which is also defined by the air in the joint cavity. The shadows of the thickened capsule (Y) at the upper part of the joint and of the ligamentum teres are also seen.

femoral head to the margins of a small acetabulum situated at the normal level. Above the acetabulum the outer surface of the capsule is intimately attached to the perichondrium of the ilium and the fascia covering the reflected head of the rectus femoris muscle.

The acetabulum is a sharply defined rounded hollow, only $\frac{1}{2}$ in. in diameter, filled mainly with soft pinkish fat lying in the expanded base of the ligamentum teres. The ligamentum teres spreads out immediately into a flattened band running upwards and outwards over the head of the femur to the fovea capitis. The head is considerably distorted from its normal spherical form, but shows no definite abnormal facets, as the child had not taken weight on the legs. The cartilage on the upper and outer surface of the head shows several small brownish pittings.

There appears to be no real articular cartilage at all on the acetabular side of the joint, but at the upper border of the acetabulum the capsule blends with the underlying cartilage of the ilium, so that in this region cartilage is forming the deep layers of the wall of the joint. This small hollow is the only socket of any kind to receive the head should a reduction be attempted. In the accompanying X-ray of the specimen (*Fig.* 416) the air which has been allowed to enter the joint spaces outlines the hollow of this small acetabulum and also the cartilage of the femoral head, and it can be seen that whereas the diameter of the acetabulum is only $\frac{1}{2}$ in., that of the femoral head is $\frac{3}{4}$ in. It is clear therefore that, even without unusual resistance from the soft parts, reduction of the dislocated head into this acetabulum is impossible. Moreover, even if it were possible to perform a partial reduction by manipulating the femoral head into a position accurately opposite the acetabulum, there is no projecting rim to keep the head in place.

AN ACCOUNT OF A CASE OF THYROID MALIGNANCY.

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THERE is now an abundant literature dealing with thyroid malignancy. The reproachful criticism of surgeons for their reluctance to study and report these cases made by Louis Wilson[1] in 1921 has been well answered by British workers, notably Dunhill and Joll. The air of mystery that surrounded these fascinating tumours has been dispelled and a simple and workable classification is now available. A correlation between the pathological findings and the clinical picture is now possible.

Joll's suggested classification[2] of the primary malignant tumours of the thyroid is as follows :—

1. Epithelial	{ a. Malignant adenoma { b. Papilliferous carcinoma { c. Carcinoma simplex
2. Connective-tissue tumours	{ a. Sarcoma { b. Endothelioma { c. Hæmangio-endothelioma
3. Mixed-celled tumours ..	{ a. Carcinoma-sarcomatodes { b. Sarcoma-carcinomatodes
4. Teratoma	

At the present day the term 'benign metastasizing goitre' is universally condemned. The forty-four cases of thyroid metastases associated with so-called normal glands or benign thyroid tumours reported by Joll in 1923 in his Hunterian Lecture on " Metastatic Bone Tumours ",[3] were reviewed in this light. Boyd[4] says of 'benign metastasizing goitre' that 'such a condition only exists in imagination'. The case reported here belongs to this group of the 'malignant adenoma'—an imperfect term, yet considered by Dunhill to be the best available at the moment.

The patient presented herself for examination at the General Hospital, Birmingham, in May, 1929, when 56 years of age—in that decade when malignant disease of the thyroid most commonly appears. A pulsating swelling was present in the right temporo-frontal region of the skull. The site of this can be seen in *Fig.* 417, which is a photograph taken a few months before death. The swelling had appeared seven months previously, and was considered by the patient to have followed an injury. The tumour was an inch in diameter, lateral to the external angular process, arising from the bone, and not attached to the overlying skin. Compression of the right common carotid artery abolished the pulsation, but did not cause any diminution in the size of the mass. The pre-operative diagnosis was 'sarcoma'.

FIRST OPERATION (May, 1929).—Mr. Percival Mills exposed an extremely vascular tumour beneath the temporal fascia. The appearances were those of a malignant growth, and after a section had been taken, radium needles (4 × 5 mgrm. and 2 × 10 mgrm.) were inserted and the wound closed. A sharp radium reaction with some signs of overdose occurred after a total irradiation of 1680 mgrm.-hours.

MICROSCOPICAL EXAMINATION OF THE SECTION.—The tissue consisted of thyroid vesicles containing colloid. There was very little deviation from the normal thyroid picture. There were areas of irregular proliferation, but the typical appearance of malignancy was not seen.

FIG. 417.—Photograph of the patient after removal of the thyroid tumour.

PROGRESS.—After the radium treatment a marked decrease in the size of the tumour occurred. No note of a thyroid enlargement was made, although the patient on her latest admission to hospital declared that a small lump had been present in the neck since she was 16. Before she left the hospital the whole skeleton was X-rayed. but no other bony tumours were detected. The only abnormality noted was a dimness of the right frontal air sinus.

The patient was re-admitted on Dec. 9, 1931, two years and seven months after her previous admission, and therefore three years after the first appearance of the skull tumour. Her general condition was good. Her chief complaint was of pain in the right ear accompanied by a little blood-stained discharge. This was diagnosed as chronic otitis media, and had been treated conservatively in the Out-patient Department. There was sign of

recurrence of the skull tumour after an apparent disappearance for over two years. More interesting still was the presence of a tumour in the left lobe of the thyroid, solid, the size of a golf ball, apparently well-encapsulated, and freely movable. The patient refused to take any interest in this tumour, and declined operation in spite of the most energetic efforts to persuade her of its danger and of its being the source of the now recurrent skull metastasis.

FIG: 418.—Photograph of cut surface of tumour. The typical appearance of a malignant adenoma. The definite capsule, the subcapsular lobules, and the central scarring are well marked.

Her stout obstinacy won the day, and the only treatment she would allow was the further implantation of radium needles into the region of the cranial tumour. A second dose of 2940 mgrm.-hours led apparently to the complete disappearance of the tumour.

She remained under out-patient supervision. The aural discharge continued and polypi appeared in the external auditory meatus. The skin over the skull metastasis ulcerated, presumably as a sequel to the radium treatment. The tumour in the left lobe of the thyroid showed no increase in size, and retained a remarkable degree of mobility.

On May 4, 1932, she was re-admitted for the third time. Mr. Stirk Adams, of the Ear, Nose, and Throat Department, reported upon her as follows : " There is an obturating polypus filling the right ear canal. The fact that with a mass so extensive there is no facial paralysis negatives a malignant disease of the middle ear, and I associate this ear infection with a grave right-sided nasal infection."

SECOND OPERATION.—After much persuasion, consent for excision of the tumour in the thyroid was obtained. The pre-operative diagnosis was 'metastasizing adenoma'. Through a collar incision with subsequent division of the infrahyoid muscles the tumour was dissected out with ease. *Fig.* 418 is a photograph of the cut surface of the tumour. The fibrous encapsulation and the central scarring, from which proceed trabeculæ enclosing areas of whitish tissue, are features invariably seen in a malignant adenoma. *Fig.* 419 represents a so-called 'adenoma' removed

FIG. 419.—So-called 'adenoma' removed from a thyrotoxic patient.

from a thyrotoxic patient, and is included for comparison with the malignant tumour.

At the same time the aural polypi were removed by the curette. The patient made a satisfactory recovery. The photograph (*see Fig.* 417) summarizes her appearance, with the healed scar in the neck, the external auditory

meatus lightly packed with cotton-wool, and the circular area of ulceration overlying the site of the skull tumour.

MICROSCOPICAL APPEARANCES.—The histology of the aural polypi provided a great surprise. Dr. F. Lamb reported as follows on the microscopical appearances :—

1. *The Aural Polypus.*—The growth has the typical appearance of thyroid gland. Only certain areas show any unusual degree of epithelial activity, and these would ordinarily be described as adenomatous. Despite its metastatic nature the growth would not be considered malignant from its histological characters alone, but this is not very unusual in the case of thyroid tumours.

2. *The Thyroid Tumour.*—The growth has the general characters of a thyroid adenoma, but is considerably more irregular in its structure and arrangement than the specimen from the ear. The cells form solid columns, and their nuclei show enlargement and irregular chromatic figures. The appearances in this specimen are much more suggestive of malignancy than are those seen in the aural polypus.

X-RAY EXAMINATION.—A second complete radiological examination of the skeleton was carried out. This revealed an erosion in the skull between the right frontal to temporal region and a marked dimness in the right frontal air cells. No secondaries in any other bones or in the lungs were seen.

SUBSEQUENT PROGRESS.—This was not satisfactory. Headache and drowsiness were accompanied by strange behaviour. A month after the operation the scar in the frontal region showed definite bulging. A rapid recurrence of the aural polypi occurred. She was discharged to the Convalescent Hospital, where her condition steadily got worse. She died suddenly on July 24, 1932, after a sudden rise of temperature of a few days' duration.

POST-MORTEM EXAMINATION.—I was able to obtain permission for a limited examination. The body was extremely emaciated. In front of the right ear a circular area of ulceration marked the site of the first noticed tumour. The right external auditory meatus was blocked by a large aural polypus. A thin supple scar was present over the region normally occupied by the left lobe of the thyroid.

The cause of death appeared to be a terminal hypostatic pneumonia. The heart showed much myocardial fibrosis with extensive atheroma of the aorta. No abnormality was detected in the abdominal viscera.

The Region of the Thyroid.—The amount of thyroid tissue dissected out represented about one-sixth of the normal gland bulk. Very little of the left lobe could be found, and was presumably composed mainly of the removed 'adenoma'. No evidence of a neoplastic process could be found. No enlargement of the cervical lymph-glands was present, and no invasion of the jugular veins was noted.

The Cranium.—On removal of the skull-cap a metastasis the size of a florin was found in the area of the right occipito-parietal suture growing from the inner table and attached to the dura. On removing the brain from the cranium a large tumour mass the size of an orange, but extremely nodular and irregular, was seen growing from the right petrous bone and indenting the temporo-sphenoidal lobe. This tumour was seen on dissection to be

continuous with the ulcerated patch in the temporo-frontal region and with the aural polypi in the external auditory meatus.

The Brain.—Apart from the intrusion of the tumour mass upon the right temporo-sphenoidal lobe, no abnormality was detected. The symptoms of increased intracranial pressure that existed in the last months of life were clearly due to the large size of the intracranial portion of the metastasis, which was the indirect cause of death. The size, nodularity, and destructiveness of the tumour quite condemn any such title as 'benign thyroid metastasis'. An extraordinary feature is that such an extensive lesion of the temporal bone could have failed to cause a complete right-sided facial paralysis.

COMMENTARY.

The case is a malignant adenoma, from which a metastasis killed the patient after it had existed for at least three and a half years. The 'adenoma' had existed since the patient was sixteen years old. An examination of

FIGS. 420, 421.—Low- and high-power views of the thyroid tumour to show area of malignant transformation and the intimacy of the cells with the thin-walled blood-vessels.

Figs. 418, 420, and 421 will reveal the changes associated with malignancy. From a central mass of fibrous tissue, trabeculæ, dense and in places calcified, run out to a true capsule, enclosing yellowish-white subcapsular areas. The arrangement may be compared with the cut surface of a pomegranate. The appearance of lobulation is striking. The strong capsule explains the free mobility of such a tumour, and its failure to infiltrate locally. Metastases occur through the blood-stream. *Figs.* 420, 421 are low- and high-power views of a portion of the tumour shown in *Fig.* 418. To the right in *Fig.* 421 a blood-vessel with an intimate lining of tumour cells can be seen.

A consideration of the photomicrographs supports the diagnosis of malignancy suggested by the macroscopic picture. The importance of examining several portions of an 'adenoma', so earnestly insisted upon by Dunhill, is well demonstrated by a glance at *Fig.* 420. The appearance of the right side of the field, so similar to that of normal thyroid vesicles, contrasts well

with the histological picture of malignant transformation seen in the left half of the photograph. The high-power view seen in *Fig.* 420 is from the latter area and shows the cell lining of the blood-vessels previously mentioned. The disorderliness of the cell arrangement is also apparent.

It will be seen, however, that a diagnosis of malignancy can be more easily arrived at by studying the macroscopic rather than microscopic structure.

The rate of malignant change was extremely slow. According to the patient's story the tumour had existed for over forty years. Metastasis occurred before any very noticeable increase in size had become obvious. Such a case must support the opinion of many surgeons that a prophylactic excision of all thyroid adenomata would lead to the disappearance of the 'malignant adenoma'. Joll considers that the presence of a metastasis does not exclude operation if the secondary can also be removed. In this case the rate of growth of the metastasis was partly controlled by radium. The discovery of another metastasis in the skull at the autopsy points to the difficulty of curing these cases once dissemination has occurred. The response to radium treatment is, however, noteworthy. The presentation of a secondary thyroid tumour as an aural polypus must be unique. The misleading nature of this has been alluded to in the case history.

I am greatly indebted to Mr. G. P. Mills, under whose care this patient was, for his permission and encouragement to publish these notes. Professor Seymour Barling kindly allowed me to use the photograph seen in *Fig.* 419, which represents a specimen removed from one of his patients.

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SHORT NOTES OF
RARE OR OBSCURE CASES

A CASE OF HYDRO-ACCESSORY-URETER IN A CHILD OF TEN YEARS.

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In view of the extreme complexity of the embryologic processes which lay the foundation of the fully developed genito-urinary system, it is not surprising that anomalies in the urinary tract are more frequent than in any other part of the body. Although a supernumerary ureter is no longer a rarity, a series of 144 cases having been described by Braasch and Scholl,[1] it is unusual for such a ureter to reach an advanced degree of hydrops and to cause misleading symptoms in a young child.

HISTORY.—Peggy B., age 10 years, was admitted to the Royal Aberdeen Hospital for Sick Children on Feb. 20, 1932. Apart from attacks of measles, whooping-cough, and chicken-pox, she had enjoyed excellent health. Occasionally she had complained of frequency of micturition. On the day before admission she was seized with acute pain over the whole abdomen. The pain was most severe immediately before micturition. She felt sick but did not vomit.

ON ADMISSION.—The child looked flushed and ill. Her pulse-rate was 144, temperature 99·2°, respirations 36.

ON EXAMINATION.—Her tongue was coated with a white fur. The abdomen did not move with respiration. Rigidity and tenderness were present over the hypogastrium, especially to the left of the middle line. Nothing could be felt in the abdomen, renal regions, or per rectum, and a specimen of urine disclosed no abnormality. The heart- and breath-sounds were normal. The clinical picture suggested acute appendicitis with pelvic peritonitis, and preparations were made for immediate operation.

OPERATION.—When the abdomen had been opened through a right paramedian incision, a small quantity of serous fluid welled up from the pelvis. The appendix, which did not appear inflamed or sufficient to account for her symptoms, was removed, and a further search instituted for the cause of the peritoneal reaction. On exploring the pelvis a round mass was found in the region of the left broad ligament. At first this was thought to be an inter-ligamentary cyst, but, on incising the peritoneum over the swelling, it was found to extend upwards into the left loin and downwards towards the vagina. It was cystic and shaped like a large German sausage. The left ureter was adherent to its posterior wall. The fact that this peculiar tumour

FIG. 422.—Accessory ureter after removal.

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lay parallel to the ureter but crossed mesial to it to reach the vagina suggested a supernumerary ureter in a state of hydrops, for Kelly[2] has shown that the upper ureter always crosses medianwards to the orifice of the lower ureter. After the descending colon had been mobilized the cystic mass was found to extend from the inner side of the upper pole of the left kidney above to the vagina below. The kidney appeared perfectly normal in size and shape, and showed no sign of lobulation or subdivision. The normal or

FIG. 423.—Ureter-catheter pyelogram : right side.

lower ureter issued from a pelvis which was slightly larger than usual. Ligatures were applied above and below, and the accessory ureter (*Fig.* 422) was removed intact. It was 16 in. in length, $4\frac{1}{2}$ in. in circumference at its widest part, and was distended with yellow, turbid fluid. It did not seem necessary to bisect or remove the portion of kidney drained by the abnormal ureter. The colon was allowed to fall back into position, and the abdomen was closed in the usual manner.

SUBSEQUENT HISTORY.—The child made an uninterrupted recovery. On being questioned more closely after the operation, the mother stated that although the child had

appeared to have complete control of micturition previous to operation, she had noticed that her clothes were frequently damp and smelt strongly of urine.

On cystoscopy fourteen days later, nothing abnormal was seen in the bladder; both sides were catheterized. Ureter-catheter pyelograms showed: Right side (*Fig.* 423)—pelvis and calices normal; left side (*Fig.* 424)—mild degree of hydronephrosis with dilatation of primary calices—probably caused by the pressure of the abnormal ureter. Urea-concentration test showed: Right kidney—2·2 per cent; left kidney—2·5 per cent.

No organisms were grown from the peritoneal exudate. A specimen of fluid, withdrawn from the excised ureter, contained ammonium carbonate.

FIG. 424.—Ureter-catheter pyelogram : left side.
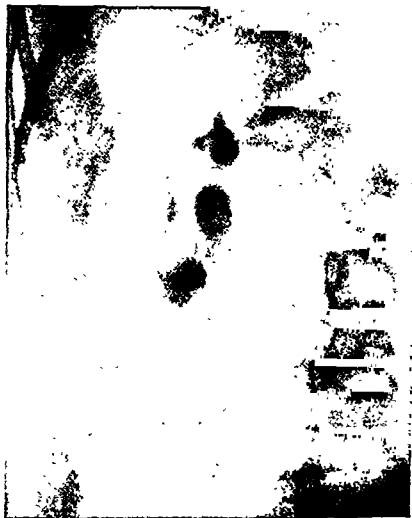
FIG. 425.—Ureter-catheter pyelogram : left side five months after operation.

0·3 per cent urea, a deposit of blood-cells and pus, but no casts or crystals. The culture was sterile.

In August the child was re-examined and was found in excellent general health. A ureter-catheter pyelogram (*Fig.* 425), taken of the left renal pelvis, showed that the hydronephrosis had almost disappeared since the accessory ureter had been excised.

DISCUSSION.

In cases of reduplication the kidney is commonly double or bifid, with the two pelves separate and always situated one above the other on the mesial surface of the kidney and each possessing fewer calices than normal. The present case corresponds more closely to one described by Huntington[3] in which one ureter terminated in a normal pelvis with the usual number of primary calices, the other in an incomplete pelvis.

The ureter develops as an outgrowth from the hinder end of the Wolffian duct near the cloaca about the fourth week. Complete duplication may result either from early splitting of the ureteral bud or from the development of two separate *anlagen*. Most frequently the ureters are found lying side by side. If the two buds come off the Wolffian duct simultaneously or nearly so, the two orifices will be found on a common ureteral ridge in the bladder, the ureter from the superior segment of the kidney terminating in the lower ureteral orifice. If a longer interval prevails, and if the Wolffian duct and ureter fail to shift anterior from the cloaca before the urorectal septum grows down to divide the rectum from the bladder, the accessory ureter may open into the rectum. If the ureter does not separate itself from the Wolffian duct but accompanies that canal caudalward, there results an abnormal connection of the ureter with those organs which arise from the duct, i.e., the vas deferens, seminal vesicle, and head of the epididymis; or else with the urogenital sinus and organs developing from this, i.e., the upper part of the urethra in both sexes and the vestibule of the vagina in women.

It would appear that a supernumerary ureter with anomalous implantation is more prone to stricture than one whose lower orifice is situated in the bladder. In the case described the gradually increasing degree of stenosis at the vaginal end probably masked the usual symptoms of incontinence and ultimately caused the extreme distension of the ureter which led to the onset of acute abdominal symptoms.

<div align="center">REFERENCES.</div>

[1] BRAASCH and SCHOLL, *Jour. of Urol.*, 1922, viii. 507.
[2] KELLY, *Diseases of the Kidneys, Ureters and Bladder*, 1914, i, 459.
[3] HUNTINGTON, quoted by YOUNG, *Practice of Urology*, 1926, ii, 39.

AN UNUSUAL COMPLICATION OF A RICHTER'S HERNIA.

<div align="center">BY J. B. G. MUIR,
TONGSHAN, NORTH CHINA.</div>

STRANGULATION of part of the circumference of the bowel, described by Richter, occurs more commonly through the femoral canal than any other hernial orifice, the sharp internal border of Gimbernat's ligament inflicting serious damage on the intestinal wall.

In a recent review on strangulated herniæ Frankau found that nearly 60 per cent of cases of Richter's hernia did not come under treatment until the condition had been present for forty-eight hours or more. The mortality is relatively high and has been quoted as being 19 per cent.

Such a hernia at the femoral canal may perforate at the site of strangulation and allow the bowel contents to escape into a pre-existing sac, which, presenting in the groin, may simulate a local abscess. The bursting of this superficially will form a fæcal fistula—or natural enterostomy. The following unusual case illustrates this sequence of events :—

A Chinese farmer, age 45, had occasionally suffered from sharp attacks

of pain in the right groin, which he found were eased by local pressure. (No tumour or swelling had ever been noticed.) Six months before admission to hospital he had suffered such an attack, but on this occasion had no relief, and after two days of great agony a tender swelling had rapidly formed in the groin. The swelling had been 'needled' by the local 'leech', and a lot of evil-smelling fluid had escaped, and also gas, with great relief of his symptoms. After a few days the local swelling and tenderness settled down, but he noticed that fæcal matter escaped from the wound several times a day, and he could only control it by a pad of wadding. No motions were passed by the rectum. Three months after the fæcal fistula had been established, he noticed a red fleshy protuberance hanging out of the fistula with an opening on the end of it, which discharged fæces. He described it as " a second penis which discharged his motions ", and from the accompanying photograph (*Fig.* 426) it will be seen that the simile is not exaggerated. He

FIG. 426.—Patient's condition on admission. Prolapsed ileum can be seen depending from the femoral canal, the lighter areas being ulceration of the mucosa. The patient had been going about in this condition for three months, all his motions being passed from the apical orifice of the prolapse.

now began to experience pain and difficulty in controlling this protrusion, and came to the Tongshan Base Hospital in June, 1932, for treatment.

It was found that a prolapse of intestinal mucous membrane had occurred from the proximal opening of a 'natural enterostomy' at the right femoral ring, the enterostomy having probably resulted from the giving way of the strangulated portion of the circumference of the bowel in a Richter's hernia. The prolapsed mucous membrane was ulcerated and hung down, completely obscuring the distal opening of the intestine, which could only be found with difficulty. All motions were being passed by the opening situated at the apex of the prolapse, and the patient stated that he had lost a 'lot of weight', and felt weak.

I decided that the only satisfactory treatment would be laparotomy with resection of the 'herniated loop', which when isolated I proposed to remove via the hernial orifice. The patient agreed to operation, and a few days were spent in cleaning up the skin around the enterostomy opening.

OPERATION (June 6).—Ether anæsthesia. The enterostomy was carefully shut off by clamping waterproof sheeting and swabs to the circumferential skin. The patient was then placed in the Trendeler.burg position, and the

abdomen was opened by a right paramedian sub-umbilical incision, with outward displacement of the right rectus muscle. A loop of ileum about two feet from the cæcum was found firmly attached to the internal aspect of the right femoral canal, the distal intestine below the loop's attachment being collapsed and much narrower than that proximal to the opening. Both the entering and returning limbs of the coil were divided about 6 in. from its attachment to the femoral canal, and both proximal and distal ends of each section were invaginated. Side-to-side entero-anastomosis was then carried out with some difficulty owing to the narrowness of the intestine distal to the hernia. This was left isolated, and the abdomen was closed. A circular incision was then made around the natural enterostomy and prolapsed ileum, and a ring of skin, together with the enterostomy and loop of ileum, was removed by easy dissection through the femoral canal (*Fig.* 427).

FIG. 427.—Prolapse and portion of ileum resected. A probe is in the apex of the prolapse. X is the proximal loop of intestine, and Y is the distal loop. The discoloured ring of excised skin was that surrounding the hernial orifice.
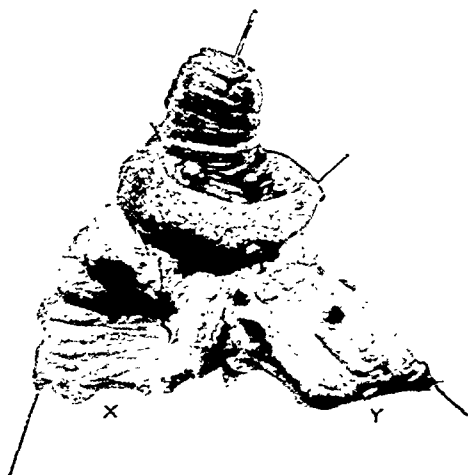
A drain was inserted down to the femoral canal, and the wound sutured. Good healing occurred, and the drain was removed on the fourth day. The first rectal motion for six months was passed on the second day. The patient made excellent progress until the third week, when some of his friends, eluding the vigilance of the nurse, smuggled some fruit into him on a visiting day. He partook of this well but not wisely, and contracted acute bacillary dysentery, from which he succumbed in three days, despite all treatment—a most disappointing end to an initially successful case.

This natural relief of a Richter's hernia by the formation of enterostomy must be somewhat rare, as also the prolapse of the mucosa from the proximal limb of the affected loop of intestine.

REFERENCE.

Frankau, C., *Brit. Jour. Surg.*, 1931, Oct., 176.

CASE OF CONGENITAL CYSTADENOMA OF THE KIDNEY.

By A. W. FAWCETT,

ASSISTANT SURGEON, ROYAL INFIRMARY, SHEFFIELD.

THE case was referred to me on account of an attack of pain and the finding of a tumour in the left hypochondriac region.   The patient, a trombone player in an orchestra, had felt the pain first whilst playing his instrument, and later found a swelling.

FIG. 428.—Pyelogram of left kidney showing filling defect at lower part of renal pelvis.

On examination the tumour was palpable and appeared to be a kidney which had apparently recently prolapsed ; it seemed to be slightly enlarged. A pyelogram was taken with the result shown in *Fig.* 428.   Chromocystoscopy revealed a slightly diminished flow of 'blue' from the left ureter.

An exploration of the left kidney was carried out, and on exposing the organ it was found to contain a rounded tumour in the lower pole. A nephrectomy was performed and the kidney hardened prior to bisection, which revealed the condition shown in *Fig.* 429—an encapsulated tumour divided into numerous polyhedral spaces containing clear fluid.

FIG. 429.—Macroscopic appearance of the kidney on bisection.

Microscopic sections show compressed remains of renal tubules in places. Dr. Harding, of the Pathological Department of the University, has kindly examined the specimen and is of the opinion that it is a congenital cystic adenoma of the kidney.

The case is of interest in two respects : (1) The cause of the prolapse was apparently partly occupational ; (2) The rarity of the condition.

TRAUMATIC RUPTURE OF BILE-DUCTS : DRAINED : CHOLEFISTULOGASTROSTOMY.

By G. H. EDINGTON,

VISITING SURGEON, WESTERN INFIRMARY, GLASGOW.

THE following case is an illustration of this serious surgical condition and of the means which were employed to effect a cure.

HISTORY.—Mr. G., age 24, civil engineer, and apparently a healthy young man, was seen with Drs. Crockett and Peill on Oct. 14, 1931. The story was that, while cycling on Sept. 16, he had a head-on collision with another cyclist. Both were on push-bicycles, and the other man was stooping over a low handle-bar. When the impact came the other man's head struck my patient in the hepatic region, and both riders were knocked off their cycles. The other man sustained injuries to his spinal column which laid him up for several months. My patient, who was motored home a distance of about

half a mile by a passing doctor, was greatly shocked. He rallied and suffered great pain in the lower abdomen, and jaundice quickly supervened.

The jaundice gradually faded during the third week, and the pain diminished so that towards the end of that week he felt able to begin some work. As the pain diminished he became conscious of a 'tightness' in the lower abdomen. This tightness gradually increased, spreading upwards towards the stomach, and a hardness was felt by the hand. Notwithstanding, he resumed work, travelling to his office by train and bus ; but he was not able to do a full day's work because of 'heaviness' or lassitude. He had no appetite and swallowed liquids only, and the increasing tightness was temporarily greatly relieved by vomiting. The vomitus was said to be yellow in colour and intensely bitter. His bowels were moved several times daily by Eno's salt ; the motions were greyish-white and soft.

On the fourth day after resuming work he strained himself while boarding a tram-car which started off suddenly. He felt a snap ' as if something had given way ' in the epigastrium. From that time there was a very rapid increase of the tightness in the upper abdomen, and epigastric swelling appeared. Co-incidently, the disappearance of the jaundice became more rapid. Two days later he had to cease work altogether, because increasing heaviness was overcoming him. After being two days at home he was sent into the nursing home on Oct. 14, 1931 (twenty-nine days after the accident).

ON EXAMINATION.—The patient was able to walk along the corridor to his bedroom. His condition on examination was : face pallid ; abdomen distended and flanks tympanitic ; liver dullness not obscured ; stomach note not very clearly obtained ; globular projection in epigastrium, fluctuant, dull on percussion, and not definitely tender. Temperature 99°, pulse 92, respiration 28. The signs suggested a collection of fluid in the lesser sac, or within the sheath of the pancreas.

OPERATIONS.—On Oct. 15 a left paramedian, supra-umbilical, exploratory laparotomy was performed. Subperitoneal fat bile-stained ; omentum presented (whether lesser or great, not determined) and was incised ; behind it was a thin-walled cyst, on opening which a torrent of dark, greenish, bile-stained fluid gushed forth. The exploring finger found a large cavity, roofed by liver on the right side ; on the left the finger could not reach the limiting wall. Cyst-wall sutured to skin, and rubber tube and iodoform gauze packing inserted into cavity. Dr. Ernest Dunlop, of the Western Infirmary Clinical Laboratory, reported that no trace of pancreatic fluid was found in the specimen.

The operation was followed by a profuse discharge of bile from the wound, and on the second day the administration of ox-bile 'Tabloids' was commenced. The gauze packing was removed on the fifth day, and the wound allowed to contract down to a fistula. Screening on the twenty-third day showed the stomach situated below the level of the fistula, and on the thirty-third day the abdomen was opened by a right paramedian laparotomy with the object of searching for and dealing with the rupture. The viscera were found to be obscured by thickened omentum through which could be felt the hard and semi-globular right lobe of the liver. Further exploration was desisted from

in order to obviate the risk of damaging the fistulous track. The fistulous orifice in the original wound was, along with a small portion of surrounding skin, then freed and the old wound opened up. The stomach was found behind non-adherent omentum. The fistulous orifice and attached skin were then anastomosed to the anterior surface of the stomach (*cholefistulogastrostomy*) and the junction covered with omentum. Both right and left laparotomy wounds were closed without drainage. The patient was allowed out of bed on Dec. 7, twenty days after the anastomosis operation, and a few days later he went home. His appetite was good and stools were normal. I did not see him again till May 6, 1932, when he came up to me at a social function. He was then so stout and well that I did not recognize him.

During the period of his residence in the nursing home, heavy night sweats and some irregularity of temperature were a marked feature of his case. He told me that this manifestation had commenced with the accident, and it continued after he left the home, with gradually diminishing severity till the middle of February, when it finally ceased.

SUBSEQUENT HISTORY.—I next saw the patient early in July, when he furnished me with the following particulars :—

He resumed business on Feb. 25, and except that he avoids lifting weights, he is quite as fit as he was before the accident. His business is largely office work and interviewing clients ; but it includes a fair amount of looking at outside work in course of construction. This entails his ascending ladders and scaffolding, going down below bridges, and wriggling into corners.

His general appearance is good, and he is alert bodily and mentally. He had lost 2 stone before his operation, but he is back to normal. He has absolutely no complaints as regards his health. He eats ordinary food well, his bowels act regularly without his requiring to take medicine, and he has no discomfort of any kind in the abdomen. Examination of the abdominal wall shows both laparotomy scars ($4\frac{1}{2} \times \frac{1}{2}$ in.) sound and without any trace of yielding. Both, however, show the coarse appearance to which the term 'keloid' is sometimes applied.

Summary.—Accident followed by partial recovery and resumption of business ; increasing distress from gradual accumulation of bile in (or around) lesser omentum ; collection drained and external fistula instituted four weeks after accident ; transplantation after thirty-second day of fistula into stomach —*cholefistulogastrostomy* ; recovery.

Unlike Mr. Waugh[1] in his recent highly interesting paper on transplantation of external biliary fistula, I had to forbear subjecting the patient to exploratory dissection, although such was my intention when I began the second operation. It is disappointing not to know the precise anatomical details of the rupture, but disappointment in that respect is counterbalanced by the happy termination of the case.

REFERENCE.

[1] *Brit. Jour. Surg.*, 1930–31, xviii. 581.

PRIMARY BENIGN NEOPLASM OF THE URETER.

By G. A. B. WALTERS,

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PRIMARY tumour of the ureter is a rare condition ; and of the published cases the majority have been malignant.[1] Melicow and Findlay,[2] reviewing the cases of simple tumour in a recent communication, found that 28 had been recorded and added one of their own.

HISTORY.—E. T., a married woman aged 58, was admitted to hospital in May, 1932. For about nine weeks she had suffered from attacks of pain in the right loin, lasting about two hours and radiating down towards the groin. The pain was never very severe or colicky in nature. For about seven weeks she had noticed a lump in the right side of the abdomen. Micturition was habitually somewhat frequent, and on one or two occasions the urine had been blood-stained. No history of previous ill health.

ON EXAMINATION.—The abdomen showed some fullness on the right side, where a large tumour could be felt in the loin ; it was smooth, mobile, and easily separated from the liver. The patient was rather thin, but except for some bad teeth she was in good condition. Blood-pressure and blood urea were well within normal limits. The urine contained a few red blood-corpuscles but showed no other abnormality. Radiological examination disclosed no evidence of calculus.

Cystoscopic examination revealed a normal bladder and left orifice ; at the right orifice a small tag of growth was presenting, and I was unable to pass a ureteric catheter on that side ; the attempt did not cause any marked hæmorrhage. After intravenous indigo-carmine there was a good excretion of dye from the left ureter in five minutes. No dye was seen from the right side in fifteen minutes. Uroselectan B showed that the pelvis and calices of the left kidney were normal ; there was no excretion visible from the right kidney.

OPERATION.—A provisional diagnosis of neoplasm of the right kidney pelvis with secondary deposit in the ureter was made, and operation performed on May 25. Under spinal anæsthesia (2½ c.c. Duracaine), a large hydronephrotic kidney was delivered with considerable difficulty, and the ureter was seen to be very tortuous and dilated ; it was divided low down with the cautery and the kidney was removed.

As owing to the dense adhesions this part of the operation had taken rather a long time, a further 2 c.c. of Duracaine were then given. Next the bladder was exposed through a sub-umbilical incision ; the right side was dissected clear, and the remainder of the ureter, together with a small wedge of bladder, including the ureteric orifice, were removed.

The bladder was drained suprapubically and through the urethra, the patient returning to the ward in good condition. With the exception of a mild attack of pyelitis convalescence was uneventful. When seen in the out-patient department three months later there was no evidence of recurrence.

PATHOLOGY.—Macroscopically the kidney showed a large hydronephrosis, little normal tissue remaining. The ureter was patent in the upper 6 in., though tortuous and dilated to a diameter of 1 in. In the lower part the lumen was occluded by growth (*Fig.* 430) as far as the orifice, through which the tumour was starting to protrude.

Examination of the section seen in *Fig.* 431 showed a simple papilloma covered with epithelium of transitional character. There was no evidence of malignancy.

FIG. 430.—Showing the growth filling the lumen of the ureter.

FIG. 431.—Greater magnification to show the structure of the tumour. (× 75.)

Commentary.—Judging from a survey of the literature this case may be regarded as presenting the more typical features of a tumour in this situation. That is to say, the growth was situated at the lower end of the right ureter, and the patient complained of renal pain, hæmaturia, and a palpable swelling. The majority of the recorded cases, however, have occurred in males.

The small amount of hæmaturia is somewhat unusual. In a number of published cases the diagnosis was suggested by profuse bleeding on attempting to pass a ureteric catheter.

It is of some interest to note the extensive procedures rendered possible by spinal anæsthesia, without undue shock to the patient. Were this form of anæsthetic not available, a two-stage operation as performed by Thomson-Walker[3] and others would probably give a greater margin of safety.

I am indebted to Mr. W. F. Neil, Honorary Surgeon to the General Hospital, Nottingham, who performed the operation, for permission to publish this case.

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[1] CHAUVIN, E., and CERATI. *Arch. d. Mal. d. Org. gen.-urin.*, 1931, v, 631.
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[3] THOMSON-WALKER, J. W., *Proc. Roy. Soc. Med.* (Sect. of Urol.), 1921, xiv, 25.

INTERNAL HERNIA THROUGH CONGENITAL APERTURE IN MESENTERY: STRANGULATION.

BY J. C. F. LLOYD WILLIAMSON,

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INTERNAL herniæ are relatively uncommon. They may be said to comprise : (1) Hernia into peritoneal fossæ, especially paraduodenal ; (2) Hernia through foramen epiploicum ; (3) Hernia through aperture in (a) omentum, (b) broad ligament, (c) falciform ligament, (d) mesocolon. (e) mesentery. Of these, probably the last type is the most rare, few cases having been recorded.

Romankevitch[1] quotes two cases of the mesenteric type as well as one mesocolic. Hamilton Bailey[2] describes a personal case of the mesenteric type, and he has assisted also at a second case : it is interesting to note that in both these cases reduction was facilitated by aspirating some of the contents of the strangulated loop. Folliasson[3] at the end of his extensive article on internal herniæ mentions the type ; he states that the hiatus is probably congenital, and usually occurs near the cæcum (as in this case). He quotes one case only, a patient of Lafourcade ; no record of the publication of this case can be found.

The present case is that of a youth of 17, who was admitted from the country at 5 a.m. on a Sunday morning complaining of very severe abdominal pain.

HISTORY.—The patient had been in normal good health till 11 p.m. the night before, when there was a rapid onset of severe generalized abdominal pain, which at times made him roll about. He vomited five or six times. The bowels had been open that day, but not after the onset of pain. Pain persisted and increased. He felt as if he wished to bring up wind, but could not. Micturition was normal. There was no history of a previous attack.

Past History. — Left orchidectomy at the age of 3, probably for tuberculosis.

ON EXAMINATION.—The patient looked grey and ill: temperature 97·4°, pulse 78, respirations 20. At intervals he was absolutely unable to be still, but rolled about in agony. The tongue was thickly furred. He pointed to the lower abdomen as the site of the pain, and palpation elicited rigidity and tenderness here, most marked in the right iliac fossa, where there was slight distension and a doughy sensation on pressure. Rectal examination showed no abnormality. The left testis was absent, the right somewhat enlarged and accompanied by a small hydrocele of the tunica vaginalis. The hernial orifices were normal. Urine showed some acetone and a considerable amount of sugar (this disappeared immediately after operation), but no albumen or organisms.

A provisional diagnosis was made of acute appendicitis, probably with a concretion giving rise to colic, and morphia and warmth were applied to the patient.

OPERATION (6.45 a.m.).—On opening the abdomen through a right lower paramedian incision, free blood-stained fluid exuded. On looking further it

was seen that 4 ft. (as measured afterwards) of the lower ileum had passed from left to right through a rounded aperture 2 in. in diameter in the distal part of the mesentery and was strangulated. The constriction was very tight, but by enlarging the hole slightly and with great care the hernia was reduced. The bowel was black, and did not recover at all, so the terminal 5½ ft. of the ileum were resected and a side-to-side ileocæcostomy was performed. The gap in the mesentery was closed, and then the abdominal wound.

SUBSEQUENT PROGRESS.—The condition was critical for the first twelve hours, but after that the patient made a rapid recovery, and now, nine months later, appears to be in good health and is gaining weight rapidly.

Comment.—Accurate diagnosis of this condition does not seem to be within the realms of possibility, but the acuteness of onset, the severity of the pain, and the vomiting should have suggested an acute internal strangulation or torsion of bowel. The origin of the aperture is obscure, but it would seem to be congenital.

I am much indebted to Mr. J. R. H. Turton for his kindness in allowing me to publish this case.

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[1] ROMANKEVITCH, V. L., "Aperture in Mesentery in Connection with Strangulation of the Intestine", *Vestnik Khir.*, 1930, xxi, 132–139 (abstr. in *Zentralorg. f. d. ges. Chir.*, 1931, liv, 199.

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AN UNUSUAL ACCIDENT IN GASTRECTOMY.

BY IAN M. ORR,

SURGEON, LONDON MISSION HOSPITAL, NEYYOOR, TRAVANCORE, S. INDIA.

AN elderly man giving a typical history of gastric carcinoma was admitted under my care, and after suitable preparatory treatment was operated upon. An infiltrating carcinoma involving the pyloric portion of the stomach was found (*Fig.* 432), and it appeared to be adherent to the pancreas but not to involve it. Glands were found along the lesser and greater curvatures, but no secondaries were found in the liver. It was considered to be a suitable case for a Pólya gastrectomy, but the adhesions to the pancreas presented a difficulty.

OPERATION.—The duodenum was divided (*Fig.* 433) and the distal end closed. After dividing off the gastrohepatic and gastrocolic omentum and exploring the relations of the growth to the lesser sac and pancreas, it was decided to cut away a portion of pancreatic tissue with the stomach, to make the eradication of the disease as complete as possible. The coronary artery was secured and glands in relation to it were dissected off, and the gastrectomy was completed in the usual manner by the Pólya method. It was then found that a duct was cut and was discharging a few drops of clear fluid. A probe was passed up and found to lead into the body of the pancreas and along the course of the main duct. As the cut end was evidently the main

duct of the pancreas, some attempt at repair was obviously indicated. A fine rubber tube 3 in. long was passed into the duct, and secured with No. 00 catgut, leaving the ends long. The stump of the duodenum was brought into relation with it without much trouble, and a slit 1 in. long made through the seromuscular coat. The seromuscular layer was then dissected up slightly
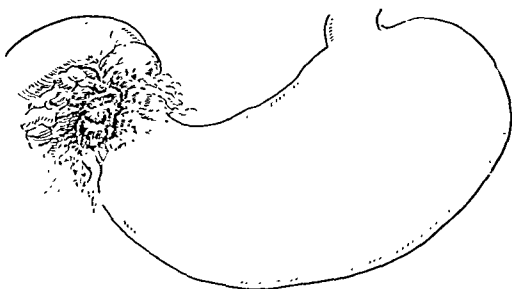
FIG. 432.—Cancerous ulcer adherent to pancreas.
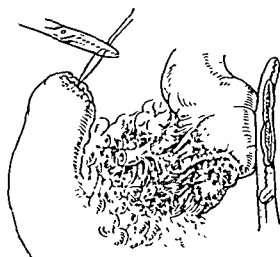
FIG. 433.—Duodenum divided and stomach turned back showing cut pancreatic duct.

on either side to form a trough, and a stab puncture made into the lumen at the distal end of the trough. Into this the rubber catheter was introduced. and the long ends of catgut from the pancreatic duct were passed by means of an intestinal needle into the lumen and out through all the coats of the bowel ¼ in. lower down. By putting traction on these two ends of catgut,

FIG. 434.—A, Duct with rubber tube inserted and trough cut in duodenal stump; B, Insertion of tube and duct; C, First layer of sutures; D, Operation completed, showing duct implanted in duodenum.

the pancreatic duct and rubber tube were drawn well into the lumen of the duodenum and secured by tying the two ends together outside the bowel. The remainder of the duct with the tube inside was caused to lie along the trough and buried by bringing the seromuscular coats together by continuous catgut suture after the manner of a jejunostomy. A second layer of suture covered all this in, and some omentum was sutured over it as an additional safeguard. (*Fig.* 434.) A cigarette drain was introduced and the abdomen closed in layers.

AFTER-TREATMENT. — The drain was removed in forty-eight hours, some serous fluid only having escaped along it. The patient was treated as for any abdominal operation.

RESULT.—He made an uninterrupted recovery and passed fully digested stools from the beginning, and left hospital well and with a moderate appetite at the end of one month, having put on weight since the operation. (The

rubber tube was never found in the motions. though the nurse was instructed to look for it.)

Comment.—Interest in the case lies in the rather unusual accident— namely, division of the main duct of the pancreas during an operation for carcinoma of the stomach. and in the fact that pancreatic juices must have drained normally into the duodenum through the new opening, and in spite of the absence of a valve action no pancreatitis developed and the patient was well at the end of one month.

TORSION OF THE GALL-BLADDER.

By JAMES F. MURRAY,

KING'S CROSS HOSPITAL, DUNDEE.

TORSION of the gall-bladder in the male is sufficiently rare to make each case worth recording. I can trace only two other published cases[1] [2] ; the other 15 cases which I have found in the literature have occurred in female patients.

HISTORY.—G. W., male, age 83 years. was seized by sudden acute pain in the right upper abdomen on June 26. 1932. This he attributed to over-indulgence in ice cream a few hours previously. The pain continued unabated
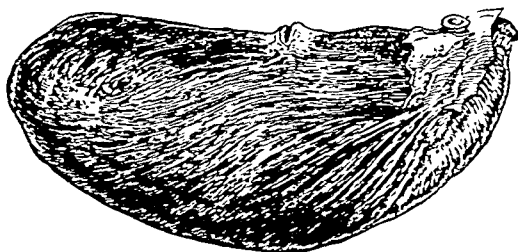
FIG. 435.—Gangrenous gall-bladder. The perforation was due to an accidental rupture while examining the organ after removal.

and was accompanied by frequent vomiting. nausea, and anorexia. Pain did not radiate or change in position. There had been no movement of the bowels. but he passed flatus freely and had no urinary disturbance. He was admitted to Dundee Royal Infirmary on June 28. 1932. There was no previous history of dyspepsia. jaundice. or colic.

ON ADMISSION.—The temperature was 98·6°. pulse 88. respiration 24, and the leucocyte count 22,000. The abdomen was retracted, respiratory movement restricted. and there was free fluid in the flanks. The epigastrium and right hypochondrium were exquisitely tender and very rigid. Liver dullness was rather increased. but owing to rigidity no abnormal mass could be felt in the right upper abdomen. A provisional diagnosis of acute chole-cystitis was made.

OPERATION.—The abdomen was opened through a right upper para-median incision. Upon opening the peritoneum a large quantity of dark

blood-stained fluid escaped and a tightly distended gangrenous gall-bladder (*Fig.* 435) immediately presented in the wound. Owing to the unusual mobility of the organ cholecystectomy was very easily performed. No stone was found in the cystic or common ducts.

PATHOLOGY.—The gall-bladder was a deep purple-black colour with thickened walls containing extravasated blood. It was tightly distended by blood and bile, but contained no gall-stones. Peritoneum surrounded it completely and was reflected off at the junction with the cystic duct. At this point the pedicle (consisting of mesentery, cystic duct, and blood-vessels) had undergone a complete clockwise torsion of one and a half turns.

SUBSEQUENT HISTORY.—The patient recovered from the operation to the extent of being up and out of bed. He then developed retention of urine, and later a severe œdema of the lung, from which he died six weeks after onset of illness.

Commentary.—The chief interest of the case lies in its occurrence in a male subject. Otherwise it corresponds closely to previous recorded cases (acute pain of sudden onset in the right upper abdomen accompanied by frequent vomiting and followed by rigidity in the right hypochondrium). In no case that I have traced was shoulder pain or jaundice noted. As in this case, a diagnosis of acute cholecystitis was usually made.

It is noticeable, too, that in nearly all cases remark has been made upon the ease with which cholecystectomy was performed by reason of the freely mobile nature of the gall-bladder.

The first recorded case was that by A. V. Wendel,[3] but in this case there was no gangrene, and perforation of the gall-bladder had occurred. By far the majority of cases occur in elderly females, and the only recorded cases I can trace as occurring in males are those noted by Costantini[1]—a male, age 42 years, who made a complete recovery, and by Holden[2]—a male age 49 years, who also recovered.

I have to thank Mr. John Anderson, D.S.O., F.R.C.S., for permission to publish this case, and Dr. D. Henderson for the reproduction of the specimen.

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SQUAMOUS-CELLED CARCINOMA OCCURRING IN A PHARYNGEAL POUCH.

BY F. C. W. CAPPS AND T. P. DUNHILL.

CARCINOMA in a pharyngeal pouch is a rare condition ; a search of the literature at the Royal Society of Medicine has failed to reveal any record of such a case ; the notes of the following case are therefore of interest.

The patient (C. P.), age 59, was first seen (by F. C. W. C.) early in May, 1932, and was admitted to the Throat Department of St. Bartholomew's Hospital on May 29. He complained of increasing difficulty in swallowing food, associated with a swelling of the left side of the neck as food was taken. Difficulty in swallowing had been present since 1902—that is, for thirty years—and he had been accustomed to 'bring back' a certain amount of food taken during the previous twenty-four hours. This gave relief until the next meal was taken. The patient also stated that his father and his father's eldest brother suffered from a similar condition, and he is said to have a female cousin of about 60 years of age still living with a pouch. This lady has not been traced, but endeavours are being made to find her. He stated that he could remember his father bringing up blood for some years before his death at the age of 75.

There was no loss of weight six weeks before admission, but at that time he brought up about 3 oz. of clotted blood, and this had happened on several occasions since. He had then started losing weight. Five days before admission he became completely obstructed and obtained no relief from vomiting.

FIG. 436.—Section of squamous-celled carcinoma in pharyngeal pouch.

A gastrostomy was performed two days later on account of the urgent need for nutrition. After feeding for nine days by means of the gastrostomy, the patient had improved sufficiently to permit a pharyngoscopic examination being made. This showed the orifice of a pharyngeal pouch almost completely blocked by a mass which was pushing up towards the orifice of the pouch from within it. Apart from the pouch and its contents, the pharyngeal wall appeared normal, but the normal lumen of the œsophagus could not be identified. A piece was nipped off through the pharyngoscope from the material in the pouch. This on examination proved to be a squamous-celled carcinoma (*Fig.* 436).

An X-ray examination carried out two years previously at the Duchess of Bedford's Hospital at Woburn showed the presence of a pouch much smaller and of regular outline both laterally and antero-posteriorly. The X-ray examination carried out by Dr. J. V. Sparks (June 4) showed the presence of a pharyngeal pouch. In the antero-posterior view (*Fig.* 437) a filling defect was seen in the middle third of the left side. A lateral film showed the filling defect to be situated anteriorly (*Fig.* 438). The growth was therefore situated in the left antero-lateral wall of the pouch, and seeing that it reached rather lower than the upper margin of the arch of the aorta, the wall of the pouch at the site of the growth was probably in contact with the left carotid artery and the junction of the internal jugular with the left innominate vein. The
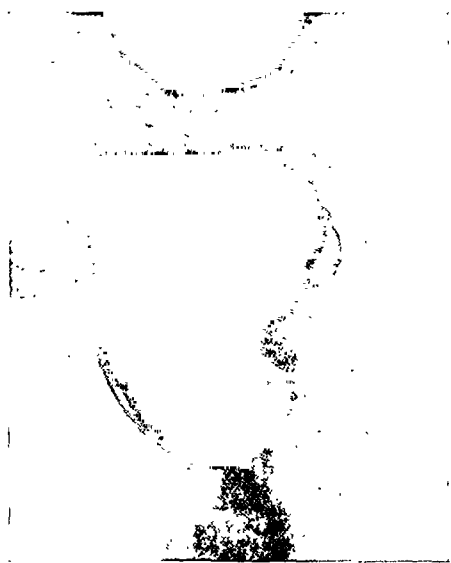
FIG. 437.—Antero-posterior view of pharyngeal pouch.
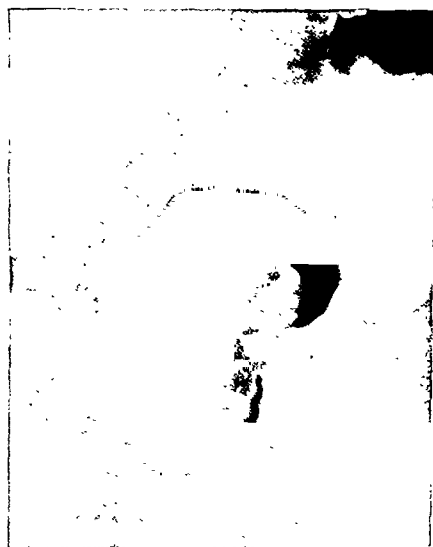
FIG. 438.—Lateral view of pharyngeal pouch.

practicability of its removal would depend on whether the growth had transgressed the wall and involved these vessels. The teeth were very bad, and a number of these were extracted on June 13.

The patient was wasted; there was an offensive smell. On attempting to swallow fluids a swelling on the left side of the neck became obvious. Pressure over this caused its contents to flow out through nostrils and mouth.

It has become my practice (T. P. D.) to operate upon simple pharyngeal pouches in one stage, although in the earlier cases the two-stage operation was employed. The one-stage operation has always gone smoothly, and the layers of the neck of the sac are much more readily recognized and more cleanly dealt with at a first operation than at a second. Naturally the patient is more comfortable with a single-stage operation. With a carcinoma in the pouch, its *growing edge close to the orifice*, and the certainty that the wall at the base of the growth must be deeply involved if not penetrated, it was

scarcely possible to consider a two-stage operation. The term 'one-stage' or 'two-stage' is used in connection with the operation upon the pouch itself; it has no reference to a preliminary gastrostomy. In a patient with a simple pouch which has become so large that swallowing is practically impossible, gain in weight and improvement in general condition follows a gastrostomy. The patient—also a patient of each of us—with the pouch illustrated in *Fig.* 439 had become greatly emaciated. Nutriment given through a gastrostomy improved his condition so much that feeding in this way was continued for five weeks and then the operation was performed without anxiety. In the present case improvement in the general condition following the gastrostomy was so slight that the risk of operation had to be undertaken without delay if the patient was to be helped.

OPERATION NOTE (June 23).—The superficial area of the neck was infiltrated with local anæsthetic solution containing 1–200,000 adrenalin. Nitrous oxide and oxygen was then given by Mr. C. L. Hewer. The sac was defined through the usual curved incision, the lower pole of the sac being freed and delivered by the gloved finger. An area about 1 in. in diameter at the base of the ulcer was thin and almost necrotic. This was attached to the origin of the carotid artery just within the upper opening of the thorax, and in separating it a little of the contents of the pouch escaped into the wound. The pouch was freed up to its neck and opened there. An œsophageal bougie was passed through the pharynx under vision into the œsophagus. The pouch was cut away by dividing

FIG. 439.—Œsophageal pouch.

the muscular layer, stripping up a cuff, and dividing the submucosa and mucosa. At one point the growth was so close to the aperture that a little of the pharyngeal wall was taken with it as a precautionary measure. The opening was sutured in two layers. The wound was packed with flavine and paraffin gauze because of the soiling.

DESCRIPTION OF THE PART REMOVED.—This was a sac measuring 5 in. by 4 in.. oval in shape, with a wall smooth on the outside, the wall of the sac being perforated at one place near the lower pole. It was filled with a friable evil-smelling growth. A section from this shows it to be a squamous-celled carcinoma, as illustrated in *Fig.* 436.

SUBSEQUENT HISTORY.—The patient remained reasonably well for eight days. when blood appeared through the dressing. This was treated by packing and pressure until it became obvious that its origin should be sought for. This was an hour after the commencement of the bleeding. The vessel, a single small artery. was quickly found and ligated. The cavity in the neck appeared clean. The patient had lost too much blood and died almost immediately.

UNUSUAL DIFFICULTIES IN THE DIAGNOSIS OF A DIAPHRAGMATIC HERNIA.

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THE readiness with which most cases of diaphragmatic hernia can be diagnosed depends on the presence in them of portions of the hollow viscera, the recognition of which by radiography is facilitated when barium is introduced into them in the form of a meal or enema. In the case here described, however, these measures failed to establish a diagnosis, and the nature of the condition remained in doubt until it was demonstrated at operation. The following are notes of the case :—

W. T., male, age 7, was admitted to the Park Hospital, Davyhulme, on Aug. 8, and again on Oct. 21, 1929. His illness commenced after a fall from his bicycle, and was characterized by abdominal pain and vomiting at the onset, mild febrile attacks, a troublesome cough, and the presence of a persistent area of dullness at the base of the left lung.

FIG. 440.—Antero-posterior view in erect position.

X-ray examination of the chest showed a semicircular opacity at the left base posteriorly. Its lower border was continuous with the shadow of the diaphragm, and its upper, convex border projected into the lung field. In addition a small abnormally translucent area was observed lying in front of the dense opacity (*Fig.* 440). It was thought that the appearances were due to diaphragmatic hernia—the opacity indicating the spleen and the translucent area representing a portion of the gas-filled splenic flexure. To confirm this view further examinations by means of an opaque enema and an opaque meal were carried out. These showed the

colon and stomach respectively to be in normal position. and we were therefore misled into doubting the original interpretation.

After his discharge from hospital in November, 1929. the patient attended from time to time for clinical and radiological observation during the ensuing twelve months. Dyspnœa on exertion. recurrent cough. and occasional pain in the abdomen and left side of the chest persisted. These symptoms were not severe, and the general health and nutrition were maintained quite satisfactorily.

On Nov. 12, 1930, the patient was re-admitted to hospital. The dullness at the base of the left lung was unchanged. Neither further X-ray investigation nor bronchoscopy threw any new light on the case. The symptoms. though mild, had persisted for more than a year and the nature of the lesion remained obscure. For these reasons an exploratory thoracotomy was undertaken on Jan. 14, 1931, under intratracheal gas-oxygen-ether anæsthesia. The eighth left rib was resected and the pleura opened. Inspection of the left cupola of the diaphragm revealed a hernia of the size of a small orange. The sac was serous and of a thickness which suggested a fusion of the peritoneum and pleura. The hernial orifice was completely surrounded by muscular fibres and was nearly circular in shape, its greatest (antero-posterior) diameter being about 2½ in. A small opening made in the summit of the sac confirmed the presence in it of the upper half of the spleen and the absence of other viscera. This opening was ligatured and the sac invaginated into the peritoneal cavity. The edges of the hernial orifice were sutured in two layers on the thoracic aspect in the antero-posterior line. Water-seal drainage was arranged by a tube through a separate opening in the tenth intercostal space. For two days after operation the patient was considerably shocked. but from the third day, in spite of a small axillary pleural effusion, he made a rapid convalescence, and he has remained well up to the time of the last examination on June 1, 1932.

DISCUSSION.

The importance of the case lies in the difficulty of interpretation of the X-ray appearances. If the nature of the lesion could have been accurately diagnosed, the symptoms produced by it would not have justified operation at the time when this was undertaken ; it would have been sufficient to keep the child under observation.

At the time of operation. at any rate. the spleen was the only viscus present in the hernial sac. This organ quite frequently forms part of the contents of a diaphragmatic sac. but reference to all the available recent literature has failed to bring to light a case in which it was present alone. The X-ray films show that the operative findings do not represent the permanent state of affairs, and further reference will be made to this point.

From the surgical point of view. the case presents one other feature of interest—the age of the patient. Truesdale[1] says that. at the time of writing. the total number of patients under ten years of age reported in the literature as operated on reached 22. with a total mortality-rate of 41 per cent. He adds another successful case of his own to the list.

Critical review of the case shows that the original explanation of the X-ray appearances was the correct one, and it is unfortunate that attempts to confirm the diagnosis by opaque meal and enema should have resulted in obscuring it. A possible explanation of the failure to demonstrate any part of either stomach or colon above the diaphragm by barium is that the small portions herniated may easily have returned to the abdomen under the weight of the opaque medium.

From a study of the series of plain X-ray films, it is clear that, while a constant volume of the spleen remained in the hernia throughout the whole period of observation, at times small portions of the stomach and colon severally occupied the sac as well (*Figs.* 441, 442).

It remains to be considered how the identity of the rounded opacity could have been established. The unusual translucency under the outer part of the left diaphragm suggests that the spleen was not in its normal position. This could have been demonstrated with certainty by inducing a pneumo-peritoneum. Such a purely diagnostic procedure is, however, not without serious danger.[2]
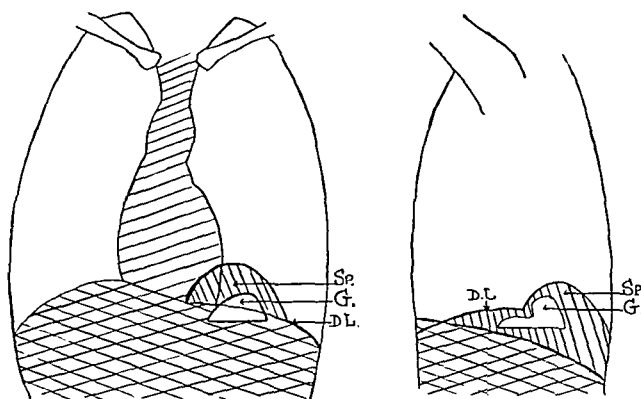
FIGS. 441, 442.—Postero-anterior and left lateral views in erect position on an occasion when a part of the stomach occupied the hernial sac as well as the spleen. Sp., Spleen; G, Gas in cardiac end of stomach; DL, Left half of diaphragm.

A test which would probably have been useful in the present case is one recently described by Benhamou, Viallet, and Marchioni.[3] According to these workers, the physiological contraction of the spleen after the subcutaneous injection of adrenalin is such as to produce a clearly recognizable diminution in the splenic shadow as observed on serial X-ray films. Mention must also be made of lienography[4]—a method of rendering the spleen opaque to X rays by the intravenous injection of thorium. This method must still be regarded as being in the experimental stage, but with the evolution of less toxic contrast media, it may in the future be available in cases of this type.

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TUMOUR OF ABERRANT LATERAL THYROID.

By FRANK FORTY,

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The following is the report of a case of tumour of an aberrant lateral thyroid.

CASE REPORT.

Miss A. C., aged 18, was admitted to the Birmingham General Hospital in March, 1932, under Mr. J. B. Leather, when she complained of a painless swelling on the right side of the neck, first noticed seven years previously. On two occasions the swelling had been aspirated, and artificial sunlight treatment had been given.

FIG. 443.—Acini lined by cubical epithelium, some containing colloid.

On Examination.—The patient was a normally healthy girl. The swelling was the size of a hen's egg, situated deep to the right sternomastoid muscle on a line with the cricoid cartilage. It was smooth, firm in consistency, and slightly lobulated. It was not tender. It could be moved freely beneath the skin, and over the underlying structures. The thyroid gland was normal.

Pre-operative Diagnosis.—Tuberculous adenitis.

Operation.—The tumour was exposed from the posterior triangle of the neck, and was dissected out without difficulty, there being no dense adhesions to neighbouring structures, and no connection with the thyroid gland. The bluish-black colour of the tumour was very striking, and recalled the appearance of a melanotic growth. It contained numerous cystic spaces with smooth walls and containing an oily greenish-black fluid.

MICROSCOPIC APPEARANCE.—Acini lined by a cuboidal epithelium are present, some containing colloid (*Fig.* 443). In places there is intra-acinar papilliferous formation (*Fig.* 444). Collections of small round cells are seen, situated chiefly beneath the capsule of the nodules (*Fig.* 445).

FIG. 444.—Intracystic papilliferous formation

FIG. 445.—Collection of lymphocytes beneath the capsule of the tumour.

POST-OPERATIVE DIAGNOSIS.—A tumour of the neck arising in lateral aberrant thyroid tissue.

DISCUSSION.

Though of relatively rare occurrence. lateral aberrant thyroid tumours form a well-defined pathological group with a growing literature. Billings and Paul[1] in 1925 found 34 recorded cases. to which they added one of their own. In 1931 Cattell[2] reported 13 cases operated upon at the Lahey Clinic, and in the same year Dunhill[3] described 4 personal cases. Interest centres chiefly around : (1) The developmental nature of the nodules ; (2) Their tendency to be the site of development of adenopapilliferous carcinoma ; (3) The possible relationship of these tumours to cystic hygroma.

Developmental Nature.—It is well known that the greater part of the thyroid gland is developed from a median diverticulum of the floor of the primitive pharynx arising at the foramen cæcum. The situation of the thyroglossal cysts in the base of the tongue and in the middle line of the neck is sufficiently explained by the course followed by the thyroid bud as it grows caudalwards to occupy its normal position in the adult.

There is good evidence that thyroid tissue also develops from the fifth pharyngeal pouch, known as the ultimo-branchial body. This rudiment descends in the lateral region of the neck on either side. to fuse eventually with the lateral lobes of the thyroid gland. though the cells later appear to atrophy and to form little or no part of the adult gland. It is difficult to avoid the conclusion that tumours containing thyroid tissue found in the lateral regions of the neck have arisen in fœtal rests associated with an arrest of this process.

The researches of Williamson and Pearse[4] lead them to the conclusion that two distinct lymph systems drain the thyroid gland : (1) The 'lymphæ comites' of the veins, which drain to the lymphatic glands of the neck ; and (2) The 'thyro-thymic' lymph system. In brief, they state that the latter is a closed lymphatic system, consisting of intra- and extra-thyroidal portions. The intra-thyroidal portion consists of lymph sinusoids draining into intra-lobular lymphatics which emerge from the gland at the hilum of each lobe. The extra-thyroidal portion consists of lymphatic channels. proceeding thence to their termination in thymic tissue. In the human embryo at a certain stage of development the thyro-thymic lymph system is a large cystic cavity pervading the structures of the neck, from the base of the skull to the mediastinum. Williamson and Pearse interpret the distribution of thyroid cancers anywhere in this region as arising within this thyro-thymic system. which they regard as one apparatus, developmentally and functionally.

Relationship to Papilliferous Adenocarcinoma.—Intracystic papilliferous change is almost universal in lateral aberrant thyroids. a feature in respect of which the case here reported conforms to type (*see Fig.* 444). Further-more. most writers agree that there is a marked tendency for this to become malignant. In support of this view it is of interest to note that of the two cases reported by Dunhill[3] which he was able to follow up, one had involve-ment of the recurrent laryngeal nerve with paralysis of a vocal cord and ultimately died of malignant disease of the pelvis, while the other died of pressure on the trachea due to local recurrence after the removal of six nodules followed by deep X-ray therapy.

On the other hand, none of the cases reported from the Lahey Clinic showed any recurrence during periods ranging from ten months to five years following local removal and post-operative Roentgen therapy. Cattell[2] emphasizes the low grade of malignancy of these tumours, and states that a good prognosis can be given following the treatment indicated, even though microscopical examination reveals adenocarcinomatous changes.

Relationship to Cystic Hygroma.—Williamson and Pearse, in the communication quoted above, regard it as probable that the cystic hygromas are due to developmental abnormalities of the thyro-thymic lymph tract. They describe these as swellings formed by endothelial-lined cysts, peculiar in that their walls are constantly found to contain lymphocytes, and recalling in this respect the thymic lymph spaces of animals. This aggregation of lymphoid tissue immediately under the capsule is similarly a constant feature of lateral aberrant thyroids (*see Fig.* 445).

A further point of interest is that the cystic hygromas tend to manifest themselves, or to enlarge, shortly after birth and again at or about puberty. These are known to be periods of enhanced activity of the thyroid gland which probably induces an increased flow in the thyro-thymic lymph system.

Clinical Features.—A correct pre-operative diagnosis appears rarely to be made, the condition being usually mistaken for tuberculosis of the cervical glands, or for one or other of the commoner swellings of the neck. Suggestive features, however, are the appearance of the tumours at or about puberty, a long history without any considerable change in the condition, the situation of the tumour beneath the sternomastoid at any point from the base of the skull to the clavicle, the usually unilateral distribution, the absence of fibrosis around and fixity of the swelling, and the total absence of local or systemic symptoms, notably of any evidence of thyro-toxicosis. At operation the bluish-black colour of the tumour appears to be quite diagnostic.

I am indebted to Mr. J. B. Leather for permission to submit this case for publication, and to Professor J. Haswell Wilson, of the University of Birmingham, for help and advice with the histology.

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REVIEWS AND NOTICES OF BOOKS.

A System of Surgery. Edited by C. C. Choyce, C.M.G., C.B.E., B.Sc., M.D., F.R.C.S., Professor of Surgery in the University of London, etc. Pathological Editor, J. Martin Beattie, M.A., M.D., C.M., M.R.C.S., Professor of Bacteriology in the University of Liverpool, etc. Third edition. In three volumes. Medium 8vo. Vol. I, pp. 1112 – xxiv, with 33 colour and 67 half-tone plates, and 285 illustrations in the text. Vol. II, pp. 1111 + xvi, with 16 colour and 11 half-tone plates and 367 illustrations in the text. Vol. III, pp. 1107 + xvi, with 11 colour and 39 half-tone plates and 277 illustrations in the text. 1932. London : Cassell & Co. Ltd. £6 net the three volumes.

It is perhaps no exaggeration to say that this work represents the most important treatise on surgery extant in Great Britain at the present moment. This does not necessarily imply that it is the best ; on this point readers must judge for themselves. But it does mean that the fifty-six surgeons who have collaborated with the editors include the great majority of the leaders of surgical thought and teaching in this country. The last edition was published in 1923, and it is rather remarkable that it has been found possible to keep the work within its present size. It actually contains about one hundred more pages, ten more plates, and a slightly increased number of text illustrations. In regard to the authors, it may be noted that these come from all over the country, Scotland and the provincial universities being well represented. Among the new authors may be mentioned the following : Birkett (radium), Dunhill (neck), Jefferson (nerves), Henry (animal parasites), Souttar (œsophagus), Walton (stomach and duodenum), Wilkie (tongue), and Julian Taylor (brain and spinal cord).

The first volume does not show any very marked changes from the earlier edition. It is concerned chiefly with the general surgical problems, such as bacteriology, inflammation, gangrene, tumours, infection, and the diseases of the breast. We are inclined to question whether the special section devoted to X-ray diagnosis is really worth separate treatment. We would suggest that the information contained in it would be better if divided up among the other contents of the book.

Pannett, writing on ulceration, commends very highly the use of elastoplast bandages in the treatment of chronic ulcers. Nitch gives a very good account of the varieties of pathology and treatment of gangrene, discussing both the vascular and neurological factors in its etiology. The section on gas gangrene gives the best account we know of the differentiation of this condition according to its extent and treatment. This is divided into four categories—namely, the local, group, segmental, and fulminating. Thrombo-angiitis obliterans is discussed in detail, and a very fair account given of the scope of ganglionectomy in its treatment. The only omission we have been able to find in this extraordinarily good section is that of puerperal gangrene.

The tannic acid method of treating burns is carefully described and well illustrated by Gardham, and it is clear that in the opinion of the majority of English surgeons this method has superseded all others in the treatment of burns which do not destroy the whole depth of the skin. There is also a full account of burns by radium and X rays.

In the last edition we were told by Martin that passive immunization as a treatment for septicæmia was still on trial. This phrase still remains ten years later. The use of the direct injection of antiseptics is dismissed in two lines and there is no mention of perchloride or mercurochrome. The section on tumours, originally written by Raymond Johnson and now revised by Lawrence, does not show so many alterations or additions as we should have anticipated. We think that the description of myeloma as a benign growth, showing no tendency to cause deposits in lymph-glands or other metastases, is probably too dogmatic, as

a considerable proportion of cases followed up in the *American Sarcoma Register* do show metastatic growths. Eloquent testimony is afforded of the very little real progress that has been made in the elucidation of cancer problems by the fact that one page suffices to summarize the work on artificially produced tumours.

The sarcomata are still classified as endosteal and periosteal, whilst the osteo-genetic type is not mentioned. This may be only a question of names, but more serious is the very scanty reference to Ewing's sarcoma. It is not referred to in the section on tumours, and in a later article in Volume III it is only given seven lines, and its remarkable resemblance to osteomyelitis and its radiosensitivity are not mentioned.

Birkett's article on radium is disappointingly short. He gives full prominence to the variation in radiosensitivity of the different cell types of epithelial growths. The problems of the bomb are not discussed.

The article by Harrison on syphilis is quite a classic. We find with regret that the problem of treatment seems to have become longer and more difficult than ever, as bismuth is now added to the mercury, iodide, and arsenic as a part of the routine method. Swift-Joly deals with gonorrhœa, and there is a good new plate showing the typical appearances found in urethroscopy.

There are no essential changes in Handley's article on the breast. In the treatment of cancer, however, he advocates the use of the diathermy knife for excision, and he refers briefly to the possibility of treating even early cases by radium, though he advises that the older and tried methods of excision should not be abandoned.

The second volume is chiefly concerned with diseases of the abdomen, together with those of the face, mouth, and gullet. In general terms it is unnecessary to criticize or comment upon this part of the work in any detail. It represents the accepted teaching of British surgeons, and this has not undergone much change since the appearance of the last edition.

Nitch, in describing the treatment of cleft palate, has included the operative methods of Veau and Gillies. We should have thought the time had come in which to relegate the methods of Brophy and Arbuthnot Lane to an unimportant place. The chapter on the œsophagus, originally written by Rigby, is brought up to date by Souttar, who gives some excellent illustrations of the endoscopic appearance of different types of cancer.

The chapter on diseases of the tongue, revised by Wilkie, gives due prominence to the important part played by radium in the treatment of cancer, whilst the more mutilating operations are omitted. Walton has revised the article by Sherren on the stomach and duodenum without making any striking changes in its general tenor. Maybury and Williams give a good account of appendicitis, with a reasoned statement advocating operative treatment in all but the late cases. We welcome this especially as coming from St. Thomas's Hospital, where the dangerous doctrine of doubt and delay was so much encouraged. Such a statement of common-sense view is particularly welcome at present in order to counteract the deplorable effects of certain recent utterances advocating a policy of *laissez faire*.

Clogg describes diseases of the rectum and anus. In the treatment of cancer of the rectum he only gives two radical methods—namely, perineal excision and abdo-mino-perineal ablation. Methods which preserve the anal sphincter are not discussed.

The article by Russell Howard on the prostate and male genital organs is full and clear. We do not think it gives quite a fair account of the possibilities and results of orchidopexy.

The section on the female genital organs is by Bonney, who still advocates the radical removal of the cervix for carcinoma rather than the use of radium. The dscription of endometrioma is very short and incomplete, giving no idea of the clinical picture of this disease when it spreads to the intestine.

We think it rather unfortunate that the article on the heart and blood-vessels by Rock Carling has had to be placed so far away from that which describes gangrene. In neither of these articles does the subject of embolectomy receive the consideration that it merits.

Volume III shows two important changes of authorship. The section on the

neck, including the thyroid and parathyroid, is written by Dunhill, and that on the nerves by Geoffrey Jefferson.

Dunhill's article on goitre is remarkable for its clear exposition of the subject and its simple classification. All conditions of hyperthyroidism are grouped together under the term 'toxic goitre '. Exophthalmic goitre is the primary form. The early secondary form presents a tumour of the neck before the symptoms. The late secondary form includes toxic adenoma, and in this the cardiac symptoms predominate.

Jefferson deals with the peripheral nervous system. He admits the experimental evidence of successful nerve-grafting, but considers that at present there is no clinical application for this method. Surgery of the sympathetic nervous system is rather briefly summarized, and further elaboration in the description and illustration of operative methods would be acceptable. The chapters dealing with the brain and spinal cord are still by Trotter, but with the assistance of Julian Taylor. For the neurological aspect of this subject we have nothing but admiration, but it is unfortunate that the surgery of the vertebral column is so very sketchy. The treatment of certain types of fractured spine from a practical point of view is more important than the theory of the nature of concussion. The fixation of fractures of the spine which are uncomplicated by spinal-cord injury require detailed and dogmatic description.

Diseases of the bone are dealt with by Choyce, who has adopted the comparatively conservative methods of Starr in the treatment of osteomyelitis. The value of Winnett Orr's packing with vaseline and fixing with plaster is recognized and recommended. In a later article by the same author on diseases of the joints we think that some mention might have been made of osteochondritis dissecans as one of the varieties of internal derangement of the knee.

The article on fractures is by Hey Groves, who in addition to the material in the last edition gives an illustrated account of the treatment of a fractured patella by means of a strip of fascia lata. We think that this article would have been more complete if reference had been made to the skin-tight plaster methods of Böhler. the advantages of operating under local anæsthesia, and the forcible correction of fractures of the os calcis.

A Synopsis of Surgical Anatomy. By ALEXANDER LEE McGREGOR, M.Ch. (Edin.), F.R.C.S. (Eng.), Lecturer on Surgical Anatomy, University of Witwatersrand, etc.; with a Foreword by Sir HAROLD J. STILES, K.B.E., F.R.C.S. (Edin.). Crown 8vo. Pp. 609 + xvi, with 606 illustrations. 1932. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

IT does not require a very long perusal of this book to make one realize that it is something fresh and unusual. One is accustomed to think of anatomy and even surgical anatomy as being something terribly dry and stereotyped. Certainly this book is neither the one nor the other. In the variety of its subject-matter and the cleverness of the illustrations it intrigues us, and we feel quite the excitement of turning over the pages of the latest thriller to see what the next chapter is going to be. Six hundred closely printed pages and over six hundred illustrations indicate something of the length of the book, but give no hint as to its features of variety and novelty. A Foreward by Sir Harold Stiles and an Introduction by Professor Dart indicate the high ideals aimed at in the book.

The two happy features of the work are the way in which the author has brought in all manner of surgical subjects and talked about them from an anatomical point of view, and also the clever way in which the artist has given diagrammatic representation of the matters described in the text.

Some of the subjects discussed are as follows, and these will give an idea of the scope of the work : the parathyroid and fibrocystic disease, the variations of the bile-ducts and cystic artery, the anatomy of the child, the sympathetic nervous system, the diaphragms of the body, the accessory bones, errors of development. diverticula. bodily habitus. the pathology of bone in terms of anatomy, the anatomical bases of clinical tests, backache, and the anatomy of surgical approach.

We have great pleasure in congratulating the author. the artist, and the publishers on the production of a very useful and remarkable book.

A Short Practice of Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern
    Hospital, etc.; and R. J. McNEILL LOVE, M.S. (Lond.), F.R.C.S., Surgeon, Royal
    Northern and Metropolitan Hospitals, etc. Two volumes. Demy 8vo. Vol. I, pp.
    536 + viii, with 269 illustrations. Vol. II, pp. 475 + viii, with 352 illustrations.
    1932. London : H. K. Lewis & Co. Ltd. 20s. net each.

IN Mr. Hamilton Bailey's former books, one always found concise statements, a
wealth of excellent illustrations, clear sketchy diagrams, and a mass of practical
information of the greatest use to students reading for examinations, and to
practitioners anxious to learn quickly a precise method to employ in any particular
condition. In this, his latest work, in which he has collaborated with Mr. McNeill
Love, one notices the same clear-cut style and the same brilliant illustrations. The
Short Practice of Surgery will undoubtedly be a popular work with students, but it
is something more than a mere text-book. The authors have successfully eliminated
all obsolete methods and introduced many modern procedures. It is an easy book
to read, and has the further advantage of differential type, the smaller being used
for rare and unimportant matter. For examination purposes we believe the book
will be widely read. The danger for examinees lies in slavishly copying its pithy
sentences, its diagrams, and its lists. A student would be well advised to supple-
ment its reading with a more philosophical and less dogmatic text-book.

Practical Anatomy by Six Teachers. Edited by E. P. STIBBE, F.R.C.S., Senior
    Demonstrator in Anatomy, London Hospital Medical School. Demy 8vo. Pp. 719 +
    xii, with 337 illustrations. 1932. London : Edward Arnold & Co. 30s. net.

THE general style of this volume, which is founded on a similar work by Professors
Parsons and Wright, is peculiar ; it cannot be regarded purely as a practical guide
for the dissecting-room, and it is scarcely full enough to be termed a manual of anatomy.
In a sense it combines the qualities of both, and, compared with the ordinary dis-
secting-room manual, is much easier to read and understand, as the description of
structures is more consecutive and coherent than is usual in books which describe
only what is seen at a given period of the dissection. As a general principle, anatomy
is treated in this volume as seen during a dissection, and in its arrangement and appor-
tionment of space to the different parts of the body, it follows on accepted lines.
    As is natural for a book that is the combined production of six expert anatomists,
its use will be in the dissecting-room or as a help for pre-clinical anatomy examina-
tions rather than as an aid to a surgeon interested in anatomy from an operative
point of view. In this respect we feel that the authors have missed an opportunity
of writing a book on practical anatomy in which the clinical importance of medicine
and surgery should be constantly in evidence. Wishing to discover whether the
authors had this point of view, we looked up the description of the abdominal sym-
pathetic and of the lymphatic drainage of the large intestine. The authors must
be aware of the importance in medicine and surgery of the sympathetic nervous
system, and no doubt realize that if medical students are to understand the applica-
tion of surgery to it, they must have an accurate knowledge of its anatomy. The
description of the sympathetic system is very sketchy, and the lymphatic drainage
of the colon, as of other organs, is scarcely touched upon. Admittedly this volume
deals only with those things which can be seen in the dissecting-room, but the
average medical student only learns anatomy with a view to the study of medicine
and surgery ; and it is to be hoped that in the next edition some space may be
allotted to these subjects, space which may be taken from the fifty pages given to
the topographical anatomy of the brain.
    It is significant that this volume is written in what is now called the old nomen-
clature, though in places the Basle terminology is introduced in brackets. A volume
written by different authors in six chapters cannot be uniform in view-point, but so
far as is possible under the circumstances it is evident that the object of each author
has been to provide a strictly practical volume. The book seems to be singularly
free from inaccuracies and is well printed and illustrated.

La Pratique chirurgicale illustrée. By VICTOR PAUCHET. Fasc. XVIII. Super royal
    8vo. Pp. 264 + vi, with 189 illustrations. 1932. Paris : G. Doin et Cie. Fr. 65.

THE eighteenth volume of Pauchet's illustrated surgery maintains the high standard
met with in previous volumes, and commences with an article by A. Tierny on
pharyngeal abscess. Dr. Tierny recommends ligature of the external carotid artery
and internal jugular vein before opening the abscess to avoid the risks of secondary
hæmorrhage, and advises leaving the external wound unsutured.

Dr. de Butler D'Ormond writes briefly on rupture of the diaphragm and
diaphragmatic hernia, and advocates the thoracic approach. Dr. Robert Soupault
describes a method of dealing with non-malignant stricture of the lower end of the
œsophagus. He exposes the cardiac end of the œsophagus, having first turned back
the left lobe of the liver, after division of the left lateral ligament (following Grey
Turner). After enlarging the opening in the diaphragm he pulls down the œsophagus
and opens it at the lower end, dividing the muscular coat only, which he slits up
longitudinally for from 6 to 8 cm. This writer also describes an operation for excision
of a dislocated semilunar bone, for which he employs local anæsthesia to the brachical
plexus.

Pauchet writes briefly on suppurating gall-bladder and intestinal obstruction
from impacted gall-stone, and also describes his method of dealing with jejunal ulcer
by a major gastrectomy with resection anastomosis of the involved jejunal loop
followed by a new gastrojejunostomy in Y. He advocates partial colectomy for
volvulus of the sigmoid, performing a lateral anastomosis outside the abdomen and
using a Paul's tube for temporary drainage.

Dr. Dartigues writes on the Voronoff operation. He employs an inguinal
incision, pushes the testicles out through the wound, and sutures the graft to the
parietal layer of the tunica vaginalis, which is scarified after rubbing the surface of
the tunica with a swab until the vessels are exposed.

Skin-grafting for repair of ruptured perineum by Leon Imbert, nerve-grafting
for injury to the musculospiral nerve, and a plastic operation for lengthening of the
biceps by George Pascalis call for no special comment.

Dr. Masmonteil writes on operative repair of a T-shaped fracture of the lower
end of the humerus. The apparatus he employs for traction is most ingenious. He
divides the olecranon, drives a long screw through the condyles transversely, and
employs a Y-shaped plate to secure the two condylar fragments to the shaft. The
employment of a long screw to join up the olecranon seems unnecessary. He also
describes a method of screwing the shaft to the neck of the femur for fracture, and
gives an account of Hey Groves's operation for repair of crucial ligaments.

Dr. Marc Iselin has written an interesting article dealing with repair of tendons
in the hand. When a tendon is divided in the finger he makes a transverse incision
in the palm and draws the tendon out of its sheath (and does the same with the distal
end through an incision beside the terminal phalanx). He then employs the figure-
of-eight 'suture en lacet' of Cunéo for each end and re-introduces the proximal
portion of the tendon into the sheath. using a special probe inserted from the
distal end. He believes in secondary suture of the main wound. This article is
very instructive.

Dr. George Pascalis employs double lateral suture with bronze wire for fracture
of the patella, and describes a method of circular subcutaneous suture for a vertical
fracture.

Recent Advances in Anæsthesia and Analgesia. By C. LANGTON HEWER, M.B., B.S.
    (Lond.), Anæsthetist and Demonstrator in Anæsthetics. St. Bartholomew's Hospital,
    etc. Large post 8vo. Pp. 187 + viii, with 64 illustrations. 1932. London : J. & A.
    Churchill. 12s. 6d. net.

As the author rightly reminds us in his preface. this is not a text-book or a volume
from which elementary instruction may be gleaned. but is a genuine attempt to
record the modern trend. methods, and scope of anæsthesia and analgesia. In this
endeavour the author has certainly succeeded, and if *some of his descriptions of new*
methods do err on the side of brevity, he has, at any rate, appended a most complete

and useful list of references at the end of each chapter. In this way the key of a very considerable storehouse of knowledge is presented to the reader.

The chapter on resuscitation during anæsthesia is both valuable and interesting; all the most modern views and methods are described, and attention is drawn to the all-important time factor in the initiation of treatment for primary cardiac failure. Chapter III, on pre-medication, synergism, and basal narcotics, though well written and easily understood, is all too short and makes the reader wish for more. However, no fewer than thirty-four clearly marked references are appended to this short chapter, and will enable the reader to seek any further information he may require. A valuable warning is given at the end of the section of Chapter III headed 'Pernocton' : " In conclusion it should be added that if any basal narcotic has been used in a combination anæsthesia, it is essential that the anæsthetist should impress upon all those responsible that no further narcotic of any description whatever should be given after operation until the patient is completely conscious and complaining of pain or is very restless. Neglect of this precaution has led to several fatalities."

Three chapters are devoted to analgesia, and are headed (1) Local analgesia ; (2) Recent advances in the technique of local analgesia ; and (3) The present position of spinal analgesia. The author is very definite in his views about the desirability of employing some general agent to abolish consciousness in addition to the use of analgesia. " A visit to a Continental clinic where no general anæsthesia is used, leaves no doubt in the mind of the visitor of the reality of 'psychic shock', although the actual technique may be of a very high order of excellence. The mentality of the patient must always be considered before deciding upon a local method. It will be found that patients can be roughly divided into two groups. On being questioned, one will say, ' I don't mind what you do to me so long as I am asleep and know nothing '. A typical example of the second type will make some remark as, ' I don't mind the operation but I do dread the anæsthetic'. The first group undoubtedly predominates, at any rate in this country, and to attempt a simple local analgesia on them is nothing short of cruelty. A basal narcotic or nitrous oxide-oxygen inhalation can usually be given as well without increasing the risk." The vast majority of British anæsthetists will agree with the author in his views upon this controversial subject.

The book is clearly printed and the illustrations are both well reproduced and really up to date. The author deserves the thanks of all interested in anæsthesia for this praiseworthy compilation of post-war advances in anæsthesia and analgesia.

Electrosurgery. By Howard A. Kelly, M.D., LL.D., F.A.C.S., and Grant E. Ward, M.D., F.A.C.S. (Baltimore). Large 8vo. Pp. 305 + xxii, with 382 illustrations. 1932. Philadelphia and London : W. B. Saunders Company. 35s. net.

In recent times high-frequency electric currents have been increasingly employed in surgery, especially in that of malignant disease, whether it be to replace the scalpel by 'electro-section', or the ligature by 'electro-coagulation', or to ensure the actual destruction of the malignant growth itself by coagulation or 'fulguration'. A study of the history of electro-surgery discloses the very large share that American investigators have taken in its progress, and the book now under review, written by two famous American surgeons, constitutes an admirable summary of the present status of this aspect of electro-therapeutics.

The physics of high-frequency currents takes first consideration, and stress is laid upon the essential differences between the 'damped' and 'undamped' wave-currents, whilst it is rightly recognized that the present tendency is towards the supersession of the 'spark-gap' apparatus by that utilizing the vacuum tube or thermionic valve. The histological changes and the physiological effects upon the tissues in general of the various types of current are fully detailed and are well illustrated by micro-photographs. Reference is next made to the general principles of electro-surgical treatment and to special points of detail in technique. Each region of the body is successively considered in relation to the possibilities and limitations

of electro-surgery, and these chapters, thoroughly up-to-date, are calculated to strike wonder in the minds of those who have not kept pace with recent electro-surgical developments. Especially interesting and instructive is the section dealing with urology, wherein is a full and well-illustrated account of the more recent methods of trans-urethral resection of contractions and 'bars' at the vesical neck and of the obstructing portions of the gland in benign prostatic hypertrophy. The subject of bladder tumours is dealt with in a most thorough manner by Hugh H. Young, who is a well-known advocate of the combination of electro-coagulation and the application of radium for such conditions, and who claims that with this dual method the majority of papillary tumours, even the malignant, can be success-fully destroyed.

This book can be strongly recommended to all progressive surgeons, and its careful perusal will serve to refute the suggestion, not infrequently made elsewhere, that the craft of surgery has now reached its limitations.

Minor Surgery of the Urinary Tract. By Hermon C. Bumpus. junr., Ph.B., M.S. in Urology, F.A.C.S., Section on Urology, the Mayo Clinic. Medium 8vo. Pp. 124, with 57 illustrations. 1932. Philadelphia and London : W. B. Saunders Company. 15s. net.

The title of this work is rather misleading, since comparatively few of the procedures described therein will be regarded, at any rate in this country, as 'minor' surgery. It treats mainly of the modern applications of endoscopic surgery—a line of practice in which the author is universally acknowledged a leading expert.

The progressive development of high-frequency endoscopic treatment is traced from its first employment by Beer in the destruction of vesical papillomata to its recent use in the trans-urethral removal of prostatic obstructions. Here it is pointed out that prostatic obstruction is caused not by the entire gland, but by only a small portion at a particular site in the urethra : it seems illogical, therefore, to perform such a major operation as a complete prostatectomy when all that is essential is the removal of the small obstructing mass from the readily accessible urethra. Out of 231 cases of prostatic hypertrophy treated surgically at the Mayo Clinic in 1931, trans-urethral resection was carried out in 82 (42 per cent). The author considers that this proportion will steadily increase, though he does not believe that prostatectomy will ever be completely displaced. The 'Bumpus' method of trans-urethral resection is described in detail.

Broders' grading of vesical carcinomata is commended, and the importance of a preliminary biopsy to ascertain the degree of malignancy is fully stressed. Con-trary to what has been stated elsewhere, this procedure has not been shown to have hastened metastasis during the seventeen years of its practice at the Mayo Clinic. The various manipulative methods for the extraction of calculi from the lower ureter are well described, and they are considered applicable nowadays to the majority of such cases.

The work is well illustrated and is fully up-to-date, as evidenced by the careful description of the uses of the ketogenic diet in infections of the urinary tract.

Tuberculose osseuse et ostéo-articulaire. By Etienne Sorrel, Chirurgien de l'Hôpital Trousseau, and Mme. Sorrel-Dejerine. Ancien Interne des Hôpitaux de Paris. Royal 4to. Pp. 514, with 640 illustrations. 1932. Paris : Masson et Cie. Fr. 350.

We have already had occasion to give an appreciative notice of former volumes in this series dealing with the radiographic appearances of the normal and pathological skeleton. The present work is again almost beyond praise, both for the beauty and wealth of illustrations and the careful detail used in discussing diagnosis and treatment. It really forms a complete treatise dealing with the subject of tuberculous disease of the bones and joints, such as will be most useful to every

orthopædist or general practitioner. The etiology and pathogenesis of the disease are given only in sufficient outline to enable the reader to understand the evolution of the morbid process, but the pathological anatomy is described and illustrated in all stages in minute and comprehensive detail. The sections on diagnosis rely very largely upon showing radiograms of other lesions which may be mistaken for tuberculosis. The treatment which is outlined is sufficient for practical purposes ; general principles of treatment are given first, followed by conservative orthopædic methods, and when necessary by methods of open operation. There are four chapters : the first deals with tuberculous disease of the bones, the second with that of the joints of the upper limb, the third with the lower limb, and the fourth with the vertebral column.

The uniform excellence of the text and the beauty of the figures do not allow of much critical comment. The description of tuberculous periostitis or spina ventosa is particularly good and complete, whilst the figures of syphilitic periostitis and osteomyelitis given for comparison are very instructive. In the sections dealing with tuberculous joints, we may perhaps take exception to the prominence given to methods of treatment which involve open excision of certain joints, e.g., the shoulder, the hip, and the tarsus. Possibly in this country we have gone too far in practically abandoning these operations, but we hesitate to accept the suggestion that excision of the head of the femur or of the astragalus can often be indicated, or that such operation will often give a lasting good functional result. In excision of the knee we do not like the use made of metallic staples and wire for fixation methods, holding them to be unnecessary and sometimes harmful. In the section on tuberculous spines, the figures of the diseased or damaged spinal cord are very complete and of high artistic merit. We think, however, that in this section, greater space might be given to illustrating the very early lesions, and less space to the late gross deformities, because the latter give no difficulty in diagnosis and allow of very little in the way of curative treatment. It is stated that sacro-iliac disease is very rare in a primary form, and therefore it is not illustrated.

As a standard book of reference this work will quickly take a recognized and leading place.

Thrombose: ihre Grundlagen und ihre Bedeutung. By Prof. Dr. A. DIETRICH (Tübingen). Pathologie und Klinik in Einzeldarstellungen. Vol. IV. Large 8vo. Pp. 102, with 26 illustrations. 1932. Berlin and Vienna : Julius Springer. Paper covers, RM. 8.20 ; bound, RM. 10.

THIS small monograph forms one of a series dealing with special problems of clinical pathology. It consists in a careful account of all the known facts concerning the incidence and accompaniments of thrombosis and embolism, with a number of well chosen illustrations showing the changes in the blood-clot and the vascular wall which are associated with these phenomena.

Clotting of the blood has been an event which has always fascinated the physiological and surgical research worker. Hunter and Lister each in his day tried to explain the mystery. In modern times the resources of biochemistry still continue to reveal new facts, but leave us still in doubt as to their meaning. Such recondite ideas as the electrical charge of the blood-platelets and the colloidal composition of the blood-plasma have been investigated, but, like the older notions of mechanical hindrances to the circulation and damage to the vessel wall, they do not provide the solution to the problem.

In regard to practical statistics, 911 cases are analysed. The proportion of cases of thrombosis which led to embolism was 53·1 per cent, and the fatal caess were 35·3. Only if two-thirds or more of the pulmonary circulation is blocked, does embolism cause immediate death. Only about one-half of the cases of pulmonary embolus gave a typical clinical picture.

The monograph concludes by a lengthy list of references to the literature, which is, however, taken solely from German sources.

Intracranial Suppuration. By E. MILES ATKINSON, M.B., B.S.(Lond.), F.R.C.S., Surgeon-in-Charge of the Ear and Throat Department, Royal United Hospital, Bath. Modern Treatment Series. Crown 8vo. Pp. 127. 1932. London : Jonathan Cape Ltd. 5s. net.

THIS little book presents in concise and reasonable form the essentials of the present day opinions regarding the pathology, symptomatology, and treatment of intra-cranial suppuration. It should prove of great use not only to the general practi-tioner, for whom it is primarily written, but also to otologists, whose acquaintance with the surgery of the brain is often restricted and vague. As a summary for the use of students preparing for the higher examinations it should prove most valuable.

When the author's views differ from those held by other surgeons—as they do here and there—good reasons are given for his opinions, a fact which adds to the interest and value of the book.

The only obvious point of criticism is the omission of any reference to the shelling out whole of the more chronic abscesses, a method of treatment which can now and then be carried out quite easily, and the results of which are far better than can be attained by any form of drainage.

Erdmann's Clinics. Excerpts selected from the Clinics of John F. Erdmann, M.D., F.A.C.S., Professor of Surgery in Colombia University. Edited by J. WILLIAM HINTON, M.D., F.A.C.S., Associate Professor of Surgery, New York Postgraduate Medical School. Large 8vo. Pp. 315 — vi, with 39 illustrations. 1932. Philadelphia and London : W. B. Saunders Company. 22s. 6d. net.

THESE abstracts from the clinics of J. F. Erdmann cover the whole field of abdominal surgery, including gynæcology and urology, as well as that of the breast, larynx, and some other regions. Together they give a clear and concise idea of Erdmann's teaching and his operative methods, and they abound in useful hints and practical suggestions which invariably bear the imprint of a most extensive personal clinical experience. Stress is laid upon the careful pre- and post-operative care of the patient, and Erdmann allows his patients up on the fifth or sixth day after an abdominal section without drainage. It is noteworthy that the post-operative radiation of 'radical' breast cases is no longer employed in the clinic unless specially requested by the patient, her physicians, or her friends, since it is considered to have increased the proportion of skin-recurrences and chest complications.

These lectures will be widely appreciated, especially by those who have visited Erdmann's clinic and have admired alike the great rapidity and the extreme thoroughness of his practical work.

The Melbourne Hospital Clinical Reports. Edited by S. O. COWEN, JULIAN SMITH, jun., et al. Vol. III. No. 1, June, 1932. Crown 4to. Pp. 58, illustrated. 1932. Melbourne : W. Ramsay (Surgical) Pty. Ltd. Annual subscription, 10s. 6d. ; single numbers, 6s.

THIS number opens by a sympathetic obituary notice by Sir Richard Stawell, K.B.E., of Dr. John Williams, who served the Melbourne Hospital from 1879 to 1904 and filled a prominent place in the early days of medical development in Melbourne. Dr. B. T. Zwar contributes an important paper on 200 cases of disease of the gall-bladder and biliary passages. He insists on the value of cholecystectomy and favours incision of the common duct when there is any doubt as to the possibility of stones being present in it. The report of a pathological demonstration by Dr. R. J. Wright-Smith on meningiomata contains some beautiful illustrations. Dr. A. E. Coates reports a case of ligature of the innominate artery for an aneurysm of the right sub-clavian artery, which he regards as traumatic in origin. He also is of opinion that the collateral circulation which developed in his case did not follow the classical course described in text-books. The volume concludes with statistics for the year 1930-1 which indicate a marked difference in the incidence of disease in Australia as compared with this country, and show also that surgical opinion in Victoria in many details does not conform to the operative practice in London.

St. Bartholomew's Hospital Reports.   Edited by Sir THOMAS HORDER, Bart., K.C.V.O., RONALD G. CANTI, WILFRED SHAW, CHARLES F. HARRIS, J. PATERSON ROSS, R. C. ELMSLIE, W. GIRLING BALL, and GEOFFREY EVANS.   Vol. LXV.   Large 8vo. Pp. 1–317, with Index of Vols. XLI–XLV.   Illustrated.   1932.   London: John Murray.   21s. net.

THE obituary notice of Sir Frederick William Andrewes recalls his charming personality and reminds those who worked within the London area during the war of the valuable work that he did in the treatment of tetanus.   Mr. Douglas Harmer's Semon Lecture on the relative value of radiotherapy in the treatment of cancer of the upper air-passages—regarded in this country as a classical authority on this question—is reprinted with admirable illustrations.   Mr. J. Paterson Ross contributes a thought-ful paper on causalgia, for which he advocates sympathectomy ; he attributes the success of this treatment to the fact that dilatation of the small vessels of the limbs disappears after operation.   Mr. R. W. Raven discusses the subject of secondary malignant disease of bone and emphasizes the incompleteness of our knowledge of this subject.

Die Lebensvorgänge im normalen Knorpel und seine Wucherung bei Akromegalie. By Professor Dr. J. ERDHEIM, A. O. Professor in the University of Vienna.   Pp. 160, with 31 illustrations.   1931.   Berlin and Vienna.   Julius Springer.

THIS book, the third of a series of special monographs on pathology and medicine, is devoted to a study of the vital processes in costal cartilage and its proliferation in acromegaly, founded on careful histological examination of the skeletons of two old women, one acromegalic and the other with a normal pituitary body.   A minute description of the development and life-history of cartilage in the normal and in the acromegalic patient is given and illustrated clearly.   The resumption of enchondral ossification of an abnormal type under the stimulus of hyperpituitarism has been thoroughly studied in the costal cartilages, and the increase in length of the ribs attributed to the resulting proliferation of cartilage ; this proliferation factor is also held responsible for the arthritic changes in acromegaly.

The author emphasizes throughout the work the harmonious relationship between the anterior lobe of the pituitary gland and body growth, and makes some tentative suggestions for the use of anterior lobe extract as a therapeutic measure.

Chirurgische Krankengymnastik.   By Dr. KARL GEBHARDT, Assistant at the Munich University Surgical Clinic, and in charge of the Sports Department.   Pp. 42, with 37 illustrations.   1931.   Leipzig : Johann Ambrosius Barth.

THIS monograph is intended to be a short introduction to medical gymnastics for doctors and masseurs, and presents a thoughtful account of some years' special experience in this work.   Emphasis is laid on the importance of accurately deciding which body tissue is injured so that treatment may be suitable ; a good account of the properties of the various tissues and the methods for the proper stimulation of each is given.   Schemes of exercises and the indications for their use are explained and well illustrated.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Radiologic Maxims. By HAROLD SWANBERG, B.Sc., M.D., F.A.C.P., Editor of *The Radiological Review.* With a Foreword by HENRY SCHMITZ, A.M., M.D., LL.D., F.A.C.P., F.A.C.S. Large 8vo. Pp. 127. 1932. Quincy, Ill.: Radiological Review Publishing Co. $1.50 net.

The 1932 Year Book of Radiology. Edited by CHARLES A. WATERS, M.D., Associate in Roentgenology, Johns Hopkins University, etc. (Diagnosis), and IRA I. KAPLAN, B.Sc., M.D., Director, Division of Cancer, Department of Hospitals, New York, etc. (Therapeutics). Medium 8vo. Pp. 750, with 498 illustrations. 1932. Chicago: The Year Book Publishers, Inc. $6.00.

Cinquante Techniques chirurgicales de Henry-Delagenière. Collected and edited by YVES DELAGENIÈRE. Large 8vo. Pp. 315, with 63 illustrations. 1933. Paris: Masson et Cie. Fr. 50.

Le Genou. Anatomie chirurgicale et radiographique: Chirurgie opératoire. By ANTOINE BASSET, Professeur agrégé à la Faculté de Paris; Chirurgien de l'Hôpital Beaujon. Royal 8vo. Pp. 189, with 120 illustrations. 1932. Paris: Masson et Cie. Fr. 45.

Final Report of the Commission on Medical Education. Medium 8vo. Pp. 560. 1932. New York: Office of the Director of Study, 630 West 168th Street.

A Short History of Surgery. By SIR D'ARCY POWER, K.B.E., F.R.C.S., Hon. Librarian of the Royal College of Surgeons of England. Crown 8vo. 1933. London: John Bale, Sons & Danielsson, Ltd. 3s. 6d. net.

Trattato di Patologia chirurgica generale e speciale. By Prof. OTTORINO UFFREDUZZI (Turin). Large 8vo. Vol. I. Pp.724 + vi, with 345 illustrations and 8 coloured plates. 1933. Turin: Unione Tipografico-editrice Torinese.

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INDEX TO VOLUME XX

ATLAS OF
PATHOLOGICAL ANATOMY

FASCICULUS VIII.

DISEASES OF THE ALIMENTARY CANAL

Compiled by E. K. MARTIN, M.S., F.R.C.S.

CONTENTS

CARCINOMA OF ŒSOPHAGUS.

(Invading Lung.)

A section through the lower third of an œsophagus and right lung.

In the lower part of the specimen is the œsophagus, the lumen of which communicates with an irregular cavity lined by ulcerating growth.

The adjacent portion of lung is gangrenous and contains a cavity which communicates with that of the carcinoma.

Pathological Museum, Leeds School of Medicine, E.120A

MICROSCOPIC STRUCTURE.—Squamous-cell carcinoma.

CLINICAL HISTORY.—The patient was admitted for severe cough. The state on admission was grave, with breathlessness. coughing. and wasting. The notes contain no reference to gastric symptoms.

AUTOPSY.—Gangrene of right lung with empyema. One subacute and four healing acute ulcers of the stomach.

S.J. SEWELL
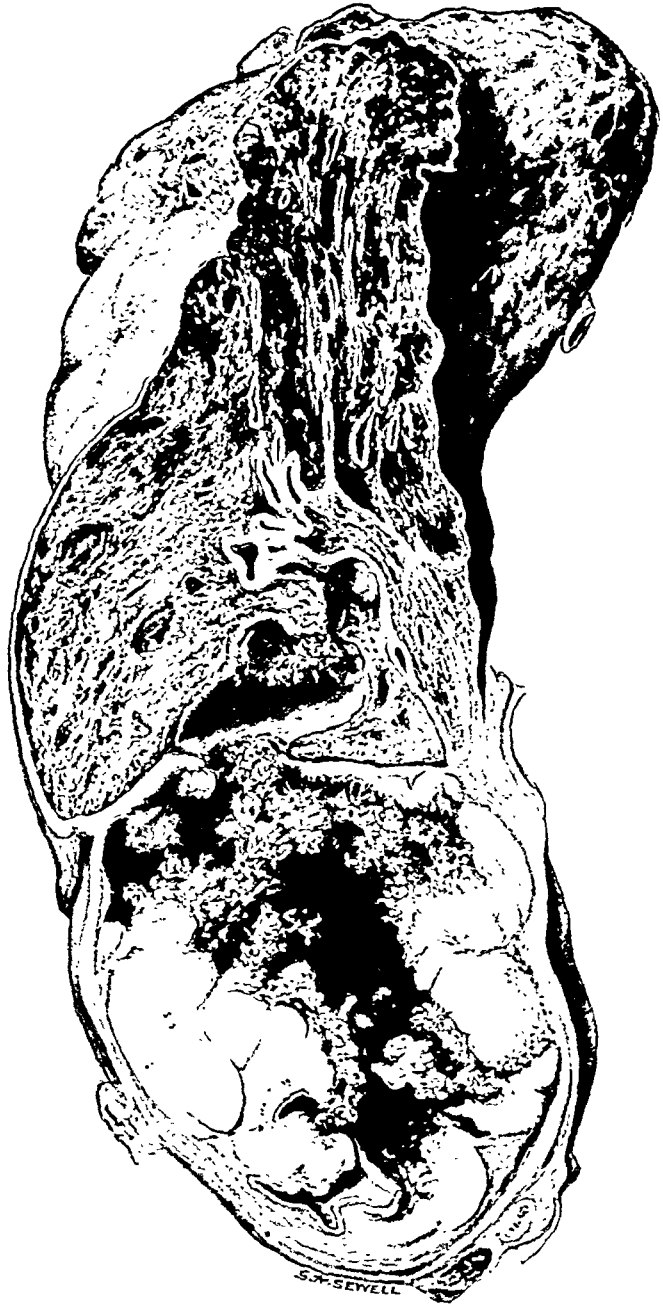
CARCINOMA OF ŒSOPHAGUS.

The lower half of an œsophagus with the cardiac end of the stomach opened from behind.

A malignant stricture of the œsophagus extends for 3 in. downwards from the level of the bifurcation of the trachea. The lumen of the œsophagus is most reduced at the upper border of the growth, which is sharply defined. Towards the cardiac side the lumen gradually opens out, and although the main bulk of the growth comes to an end abruptly a submucous infiltration extends down through the cardia and opens out into a large irregular ulcer on the anterior wall of the stomach. The perforation in the lower part of the crater was closed during life by adherent structures. The glands by the side of the œsophagus are enlarged by growth.

Museum of University College Hospital, Alim. B4

MICROSCOPIC STRUCTURE.—Squamous-cell carcinoma.

CLINICAL HISTORY.—The patient was a wasted man, aged 55, who for eight months had been unable to swallow solids and for four months had brought up food and mucus. He had vomited blood twice.

X-ray showed obstruction at the manubrio-sternal junction.

He died nine months after the onset of symptoms.

A.K.MAXWELL.

CARCINOMA OF STOMACH INVADING ŒSOPHAGUS.

The cardiac portion of a stomach with the lower end of the œsophagus.

There is a carcinoma of the cardiac end of the stomach, from the upper border of which a linear extension 4 in. in length has spread up the œsophagus. In addition, there is a circular ulcer immediately above the cardia, and above this again four minute flattened elevations which are deposits of growth along the line of the lymphatics.
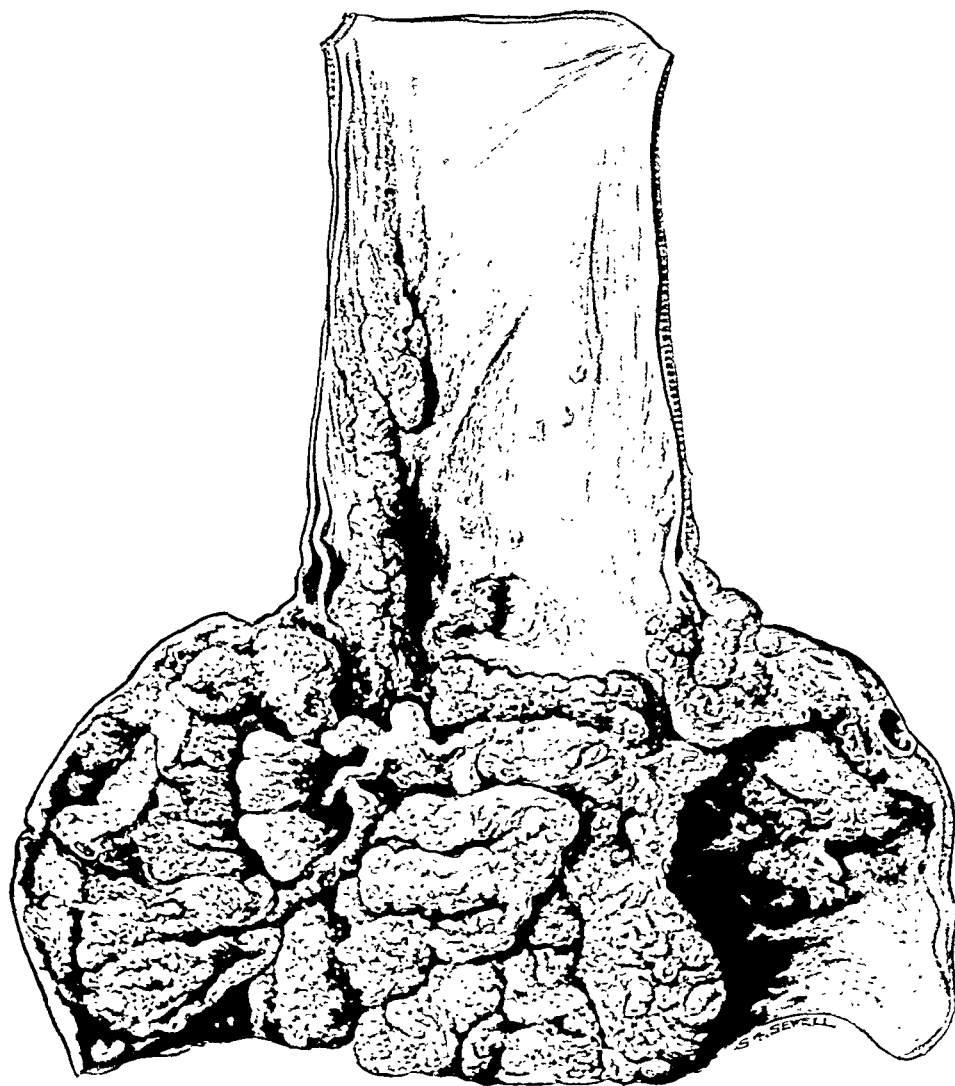
Hunterian Museum, R.C.S., 2128.1

MICROSCOPIC STRUCTURE.—Columnar-cell carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 60, who had complained of pain and vomiting after food for two years. He gradually became anæmic and emaciated. Shortly before death œdema of the legs and scrotum appeared.

AUTOPSY.—No visceral metastasis.

NO. 29—SUPPLEMENT

I 1

XLII. CARCINOMA OF THE RECTUM.

CARCINOMA of the rectum is a common disease after the age of 40 and is occasionally seen in younger subjects. It affects men about twice as often as women. Carcinoma may arise in a previously healthy rectum, but in many cases is associated with the presence of adenomata, in one of which or in the less common papilloma it may commence. The common sites of origin are the ampulla and the junction of the pelvic colon with the rectum. Carcinoma of the rectum starts in the glands of the mucosa, and is composed of columnar cells arranged to form atypical acini lying in a fibrous stroma.

The earliest appearance is that of a nodule projecting above the surface of the mucosa with infiltration of the submucous coat around its base. The further development of its naked-eye appearance is the resultant of the proliferative activity of the cancer cells and the destructive effects of infection from the contents of the bowel. When the blood-supply of the growth is abundant and its power of proliferation is vigorous a bulky tumour is produced which projects into the cavity of the rectum and may spread extensively round the bowel without much penetration of the rectal wall. More commonly, and in all cases after a variable lapse of time, progressive infiltration of the submucosa by carcinoma tends to reduce the blood-supply to the more superficial parts of the growth, which is thereby rendered less able to resist infection. The central part necroses and an excavated ulcer with raised edges is the result.

From its point of origin a carcinoma of the rectum spreads round the circumference of the bowel in the submucosa and by its bulk and by contraction of its fibrous stroma produces a stricture. A cancer of the pelvi-rectal junction has a slightly greater tendency to spread in the long axis of the rectum than one arising in the ampulla, and ultimately forms a stricture about three inches long. The tumour is often prolapsed into the lower part of the rectum as if forming the apex of a potential intussusception and so is rendered more readily palpable from below.

Penetration of the muscular coat of the rectum by a carcinoma occurs progressively but bears no constant relation to the extent of infiltration in the plane of the submucosa. When a specimen at an early stage of growth is examined in section, finger-like processes of cancer can be seen dipping down from the deep surface of the tumour between the muscle bundles. Later these coalesce, and in the stage of excavated ulcer the whole thickness of the rectal wall may be destroyed. It is probable that invasion of the lymphatic territories draining the rectum does not occur until the muscular coat has been penetrated by growth. In the submucosa the spread of cancer is always more extensive than in the mucous membrane and there is therefore a zone of infiltration which can usually be felt beyond the visible limits of the tumour as seen from the mucous surface. Microscopic infiltration in the lymphatics of the submucosa seldom extends far beyond the macroscopic edge of the growth, but occasionally a secondary nodule or ulcer is found two or three

inches above an ampullary carcinoma with apparently normal mucosa between the two. In such cases it is probable that the second growth represents an outcrop on the mucosa from a lymphatic extension in the submucous layer.

So long as a carcinoma remains confined to the rectal wall its progress is relatively slow and the prognosis after removal by operation is good, but as soon as the muscular coat is penetrated dissemination commences in the tissues outside the rectum and is usually a much more rapid process. Extension by lymphatic channels outside the rectal wall leads to secondary deposits of cancer in the glands behind the rectum and in those which lie along the course of the superior hæmorrhoidal and inferior mesenteric arteries. Owing to the inevitable infection of an ulcerating cancer through the contents of the bowel the regional lymph-glands are frequently enlarged by inflammation. For this reason microscopic examination alone can afford conclusive evidence of the presence or absence of secondary deposits of cancer in enlarged lymphatic glands. Much less commonly, and especially when the perirectal cellular tissue is infiltrated, the glands of the internal iliac chain are enlarged and lymphatic extension may, in rare cases, penetrate the levator ani and lead to secondary deposits in the fat of the ischio-rectal fossa. An interesting, though rare, type of lymphatic spread is that which involves the lymphatic plexus of the retroperitoneal tissue and shows as a rigid sheet of growth extending upwards over the posterior abdominal wall. Secondary deposits in the liver are common, and probably travel from the rectum by way of lymphatic channels, though the portal blood-stream also offers a possible route.

Direct extension from the primary growth to neighbouring structures involves the prostate, bladder, or vagina, and may lead to the formation of a recto-vesical or recto-vaginal fistula. Fistulæ may also follow invasion of the ischio-rectal fossa through the levator ani. Postero-lateral extension may involve the sciatic plexus and produce pain in the distribution of the implicated nerves. Many of the effects on surrounding structures are enhanced by the infection which accompanies the malignant infiltration.

The peritoneum on the anterior aspect of the rectum is frequently invaded by nodules of growth which may spread widely and cause ascites or obstruction of the small intestine by interference with coils which have become adherent in the pelvis. A fistula may open between such an adherent coil and the primary growth in the rectum, an accident which may be disconcerting to a patient upon whom colostomy has been performed. Perforation of an ulcerating carcinoma of the rectum into the peritoneal cavity is an occasional cause of death through a rapidly spreading peritonitis. Secondary growths in the lungs and in the skeleton are very rare.

A carcinoma of the rectum secretes a variable quantity of blood, mucus, and pus, but the amounts of these may be insufficient to attract the attention of a patient while the disease is in an operable stage.

Obstruction, though capricious in its clinical manifestations, always results in an increase in the intensity of infection of the contents of the colon. Relief from the results of absorption from the bowel above a stricture is often a striking result of colostomy. The morbid changes in the wall of the colon consequent upon obstruction of the rectum by a cancer are hypertrophy of

the muscular coats and ulceration of the mucous membrane with its attendant œdema of the whole thickness of the bowel. The ulceration is most evident immediately above the stricture and in the cæcum, which may become greatly distended. Perforation of an ulcer of the cæcum is an occasional cause of death in unrelieved malignant obstruction of the rectum.

Colloid degeneration is a common change in rectal carcinoma, but beyond increasing the bulk of the resulting tumour has no influence on its destructive power. The corresponding secondary deposits usually, but do not necessarily, show colloid change.

CARCINOMA OF RECTUM.

A rectum opened by longitudinal section.

A nodular, papilliferous tumour projects from the right lateral wall. On the reverse side of the specimen the carcinoma cannot be seen. The lymph-glands are not enlarged.

Museum of St. Mark's Hospital, 1754

MICROSCOPIC STRUCTURE.—The growth is an adenocarcinoma which has spread by direct continuity into the circular muscle coat but has not reached the longitudinal muscle. There is no extra-rectal spread and the regional lymph-glands contain no metastases.

CLINICAL HISTORY.—The patient was a man, aged 58, who complained of diarrhœa and the passage of blood and slime for $2\frac{1}{2}$ months before admission to hospital.

Sigmoidoscopy showed the tumour. The rectum was removed by perineal excision and the patient made an uninterrupted recovery.

AFTER-HISTORY.—The patient was alive and well two years later.

CARCINOMA OF RECTUM.

A longitudinal section of a rectum divided through the tumour.

The growth projects above the surface and has just commenced to invade the submucosa. There is no infiltration of the muscle and the regional lymph-glands contain no metastases.
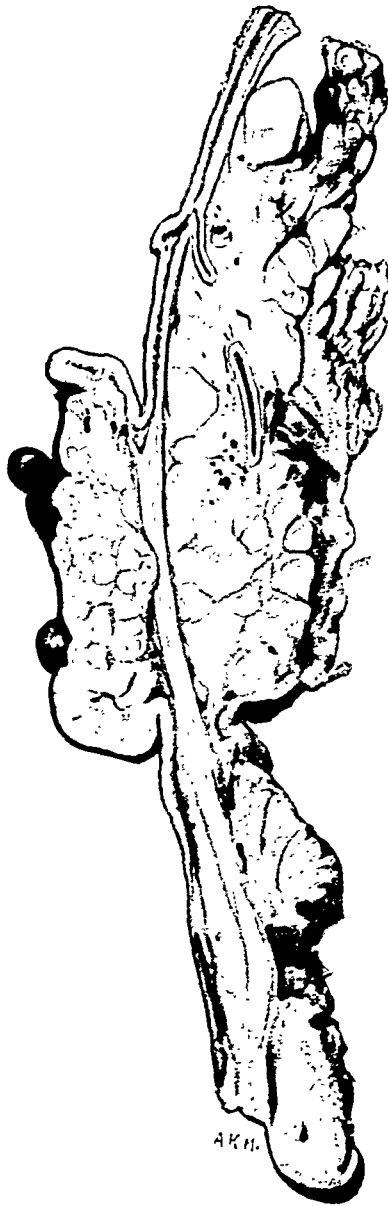
Museum of St. Mark's Hospital, 876

MICROSCOPIC STRUCTURE.—Adenocarcinoma.

CLINICAL HISTORY.—The patient was a man, aged 61, who was admitted to hospital complaining of bleeding for about nine months with no other symptoms.

The tumour was seen through the sigmoidoscope and the rectum was removed by perineal section. Recovery was uninterrupted.

AFTER-HISTORY.—Two years after operation the patient developed an enlargement of the prostate and died of uræmia. There was no evidence of recurrence.

CARCINOMA OF RECTUM.

AKM.

MUSEUM OF ST. MARK'S HOSPITAL, 876

Atlas of Pathological Anatomy

CARCINOMA OF RECTUM.

A rectum opened by longitudinal section.

On the posterior surface is a flat, shallow, ulcerating tumour. On the reverse side of the specimen the growth is seen to have penetrated the rectal wall.

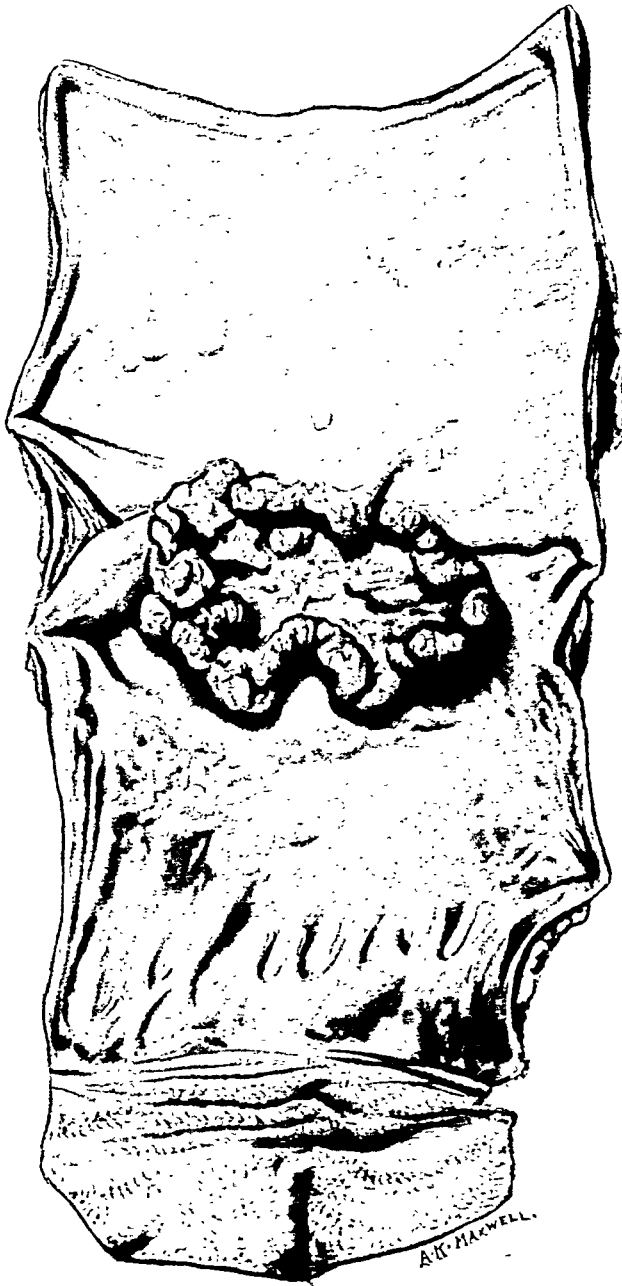*Museum of St. Mark's Hospital,* 2372

MICROSCOPIC STRUCTURE.—The growth is an adenocarcinoma which has extended by direct continuity into the extra-rectal tissues. The regional lymph-glands contain no metastases.

CLINICAL HISTORY.—The patient was a man, aged 58, who complained of irregularity in the action of the bowel for about four months before admission to hospital.

The growth was seen through the sigmoidoscope, and the rectum was removed by perineal excision. Recovery was uninterrupted.

AFTER-HISTORY.—The patient was alive and well twelve months later.

A.K. MAYWELL.

CARCINOMA OF RECTUM.

Two longitudinal slices through the wall of a rectum.

A flat, superficially ulcerated carcinoma is growing from the mucous membrane and has infiltrated the muscular coat by root-like processes which are pushing their way into the extra-rectal tissues. There were no metastases in the regional lymph-glands.

Museum of St. Mark's Hospital, 1055

MICROSCOPIC STRUCTURE.—Adenocarcinoma.

CLINICAL HISTORY.—The patient was a man, aged 50, who complained of increasing constipation and frequent desire for defæcation during the eighteen months before admission to hospital.

The growth was seen through the sigmoidoscope, and the rectum was removed by perineal excision. Convalescence was uninterrupted.

AFTER-HISTORY.—The patient was alive and well three-and-a-half years later.

CARCINOMA OF RECTUM.
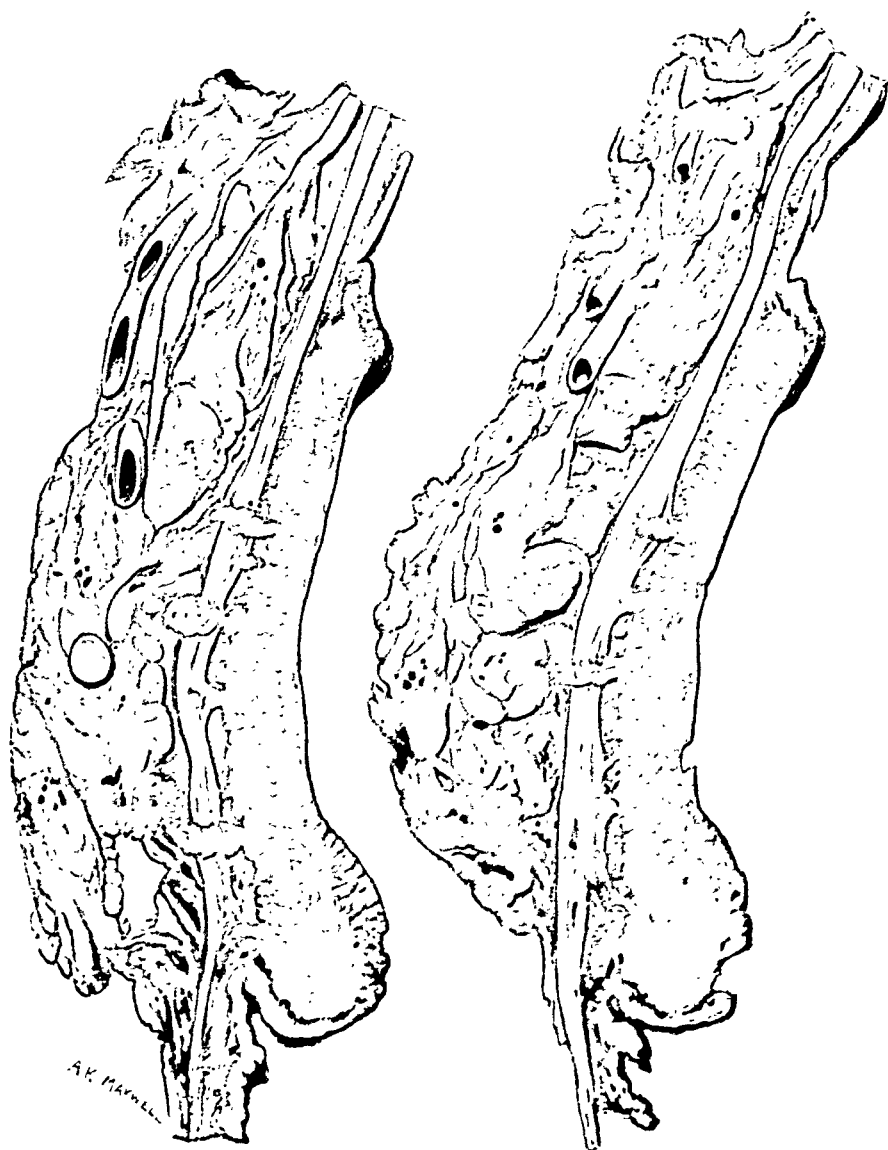
A rectum opened by longitudinal section along its anterior wall.

There is an ulcerating growth with everted edges in the ampulla. The glands on the superior hæmorrhoidal vessels contain growth.

Museum of St. Mark's Hospital, 2672

MICROSCOPIC STRUCTURE.—The tumour is an adenocarcinoma which has spread by direct continuity to the perirectal tissues. The regional lymph-glands contain metastases.

CLINICAL HISTORY.—The patient was a man, aged 64, who complained of bleeding and painful defæcation for six months. He had had loose motions for three months.

The growth was seen through the sigmoidoscope, and the rectum was removed by perineal excision. Recovery was uneventful.

AFTER-HISTORY.—The patient was alive and well six months later.

CARCINOMA OF RECTUM.

A.K.MAXWELL.

MUSEUM OF ST MARK'S HOSPITAL, 2672

Atlas of Pathological Anatomy

CARCINOMA OF RECTUM.

A rectum opened by longitudinal section.

An ulcerating growth with raised, everted edges encircles about three-quarters of its circumference.

MICROSCOPIC STRUCTURE.—The tumour is an adenocarcinoma which has spread by direct continuity into the extra-rectal tissues. Metastases are present in the regional lymph-glands.

CLINICAL HISTORY.—The patient was a man, aged 48, who complained of diarrhœa and the passage of blood for eleven months before admission to hospital.

The rectum was removed by perineal excision, and convalescence was uneventful.

AFTER-HISTORY.—He was alive and well twelve months later.

CARCINOMA OF RECTUM.

Part of a rectum opened by longitudinal section.

A pedunculated tumour springs from the mucous membrane by a pedicle ½ in. in diameter. The greater part of its surface is necrotic. On section the tumour is solid, and from the lower border of its pedicle projects a submucous extension.

Hunterian Museum, R.C.S., 1761.1

MICROSCOPIC STRUCTURE.—Columnar-celled carcinoma.

CLINICAL HISTORY.—The patient was a woman, aged 51. Six years before the onset of symptoms she had had her uterus removed together with an ovarian cyst on each side. For three months before admission to hospital she had suffered from constipation and diarrhœa and had passed blood by the rectum.

On examination there was an ulcerating growth on the anterior wall of the rectum 3 in. from the anus.

At operation the lower 8 in. of the rectum were removed with many enlarged glands.

AFTER-HISTORY.—She died nine months later of lymphatic metastasis.

CARCINOMA OF RECTUM.
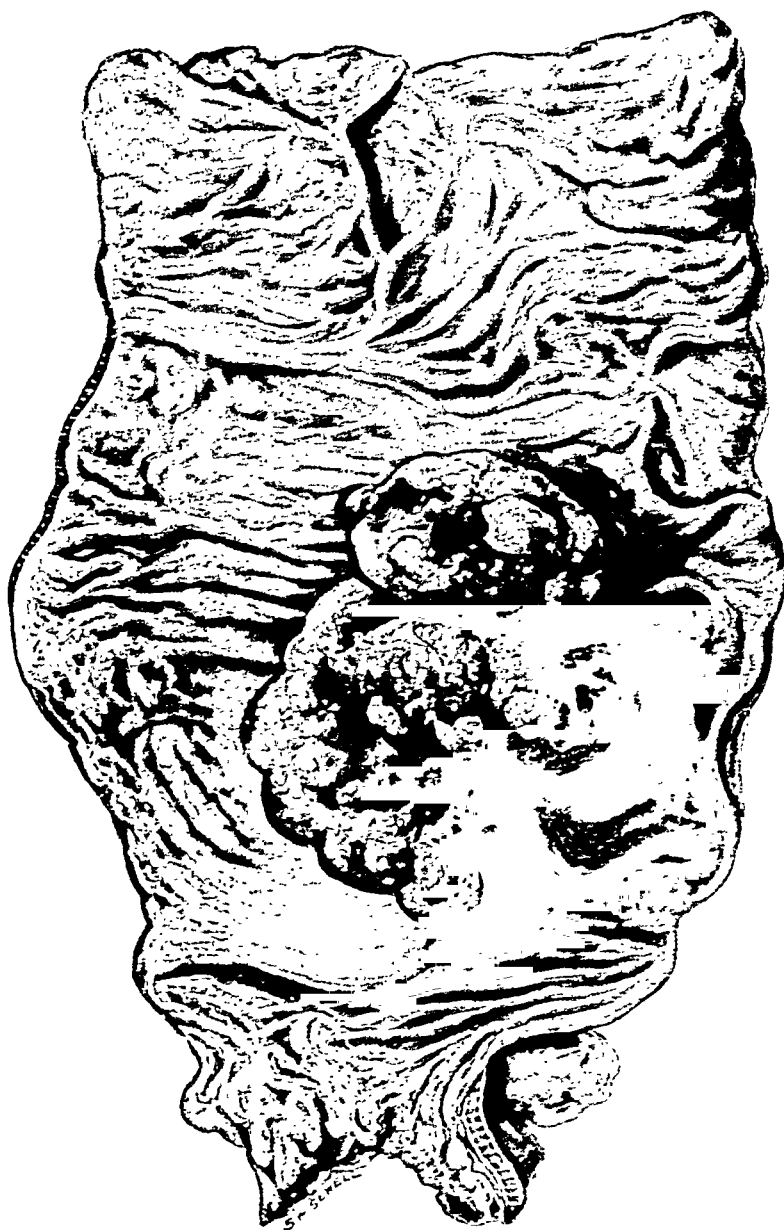
CARCINOMA OF RECTUM.

A rectum and part of a pelvic colon opened along the anterior surface

Four inches above the anus is a deep circular ulcer 1¾ in. in diameter with raised irregular edges. It extends round the whole circumference except for about half an inch. The rest of the mucous membrane is normal. On the posterior surface the inferior mesenteric artery has been dissected with the associated lymph-glands. Most of the glands are enlarged by growth.

Hunterian Museum, R.C.S., 2048.1

MICROSCOPIC STRUCTURE.—Columnar-celled carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 28, who had suffered from pain with blood in the stools for two years. There was no constipation or wasting. He had had syphilis eight years before. The specimen shown was removed by operation.

No after-history.

MUCOUS SURFACE.

BACK OF RECTUM.

The viscera of a female pelvis divided by sagittal section.

At the junction of the pelvic colon with the rectum is a constricting tumour which has replaced the whole thickness of the rectal wall and has reduced the lumen to a narrow slit.

The specimen illustrates the characteristic intussusception of the growth into the rectum below.

The tumour in the posterior wall of the uterus is a fibromyoma.

*Pathological Museum,
University of Birmingham,*
35.6 : 66.4E

MICROSCOPIC STRUCTURE. — Columnar- cell carcinoma.

No clinical history.

CARCINOMA OF RECTUM.

(ADENOMATA.)

A rectum opened by longitudinal section.

An oval, ulcerated carcinoma occupies about half the circumference of the bowel. Its lower edge is $2\frac{1}{2}$ in. above the anus. Numerous small adenomata are scattered over the surface of the mucous membrane.

Museum of St. Mark's Hospital, 665

MICROSCOPIC STRUCTURE.—Columnar-celled carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 47, who was admitted to hospital complaining of bleeding and an offensive discharge from the rectum.

The growth was seen through the proctoscope and was removed by perineal excision after colostomy. The patient made a good recovery from the operation.

AFTER-HISTORY.—Death three years and one month after operation.

CARCINOMA OF RECTUM.

A. K. MAXWELL

NO. 30—SUPPLEMENT

CARCINOMA OF RECTUM.

The lower 6 in. of a rectum, together with the anus, opened longitudinally.

A triangular ulcer, with hard margins and floor, is situated $2\frac{1}{2}$ in. above the anus. The mucosa is drawn in towards it in folds and several small sessile polypi lie around it. There is a large pedunculated polypus just above the anal canal.
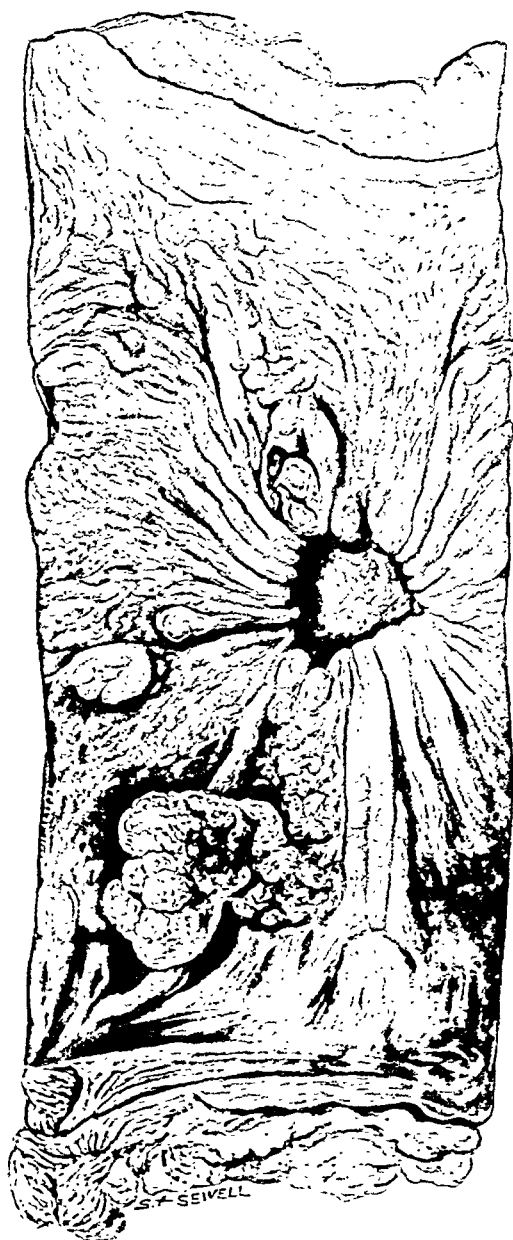
Pathological Museum, Leeds School of Medicine, 8239

MICROSCOPIC STRUCTURE.—In its deepest parts the ulcerated area shows typical adenocarcinoma, which has penetrated both submucous and muscular coats. The floor of the ulcer is covered with a thin layer of granulation tissue which contains groups of cancer cells at intervals.

The largest polypus has the usual adenomatous structure. There is some simple ulceration round its pedicle.

CLINICAL HISTORY.—The patient was a man, aged 49, who had suffered from piles for six years, but had been otherwise in good health. For nine weeks before admission to hospital he had been passing mucus per rectum. A diagnosis of carcinoma was made after digital examination, and the rectum was removed by operation. It was adherent to the prostate.

CARCINOMA OF RECTUM.

CARCINOMA OF RECTUM.

The lower $3\frac{1}{2}$ in. of a rectum opened by longitudinal section.

An annular ulcerated growth with undermined edges extends round the whole circumference of the bowel for a distance of $1\frac{1}{2}$ in., its centre being about the level of the peritoneal reflection. The lumen of the rectum is considerably diminished by the growth, which has extended through the whole thickness of the wall and is invading the perirectal tissues.

Hunterian Museum, R.C.S., 1763.1

MICROSCOPIC STRUCTURE.—Atypical columnar-celled carcinoma. The growth consists of groups of cells continuous with the glands of the mucous membrane. The cells are columnar in a few spots near the surface only. The greater part of the growth consists of spheroidal cells arranged in groups devoid of a lumen.

CLINICAL HISTORY.—The patient was a woman, aged 61. Eight years before she had been treated for what she supposed to be peritonitis. The attack was followed by recurrent pains in the left side of the abdomen. She was always constipated, but had been more so in the last few years. There was no diarrhœa, but there was a mucous discharge over which she had no control, with slight streaks of blood on several occasions. There was no rectal pain, but a feeling of weight and discomfort.

On examination the sphincter was weak. Two inches above the anus a malignant growth completely encircled the bowel. The stricture admitted the finger. The mass was freely movable. It was removed by operation at which several glands were found in the mesorectum.

She was free from recurrence one year later.

CARCINOMA OF RECTUM.
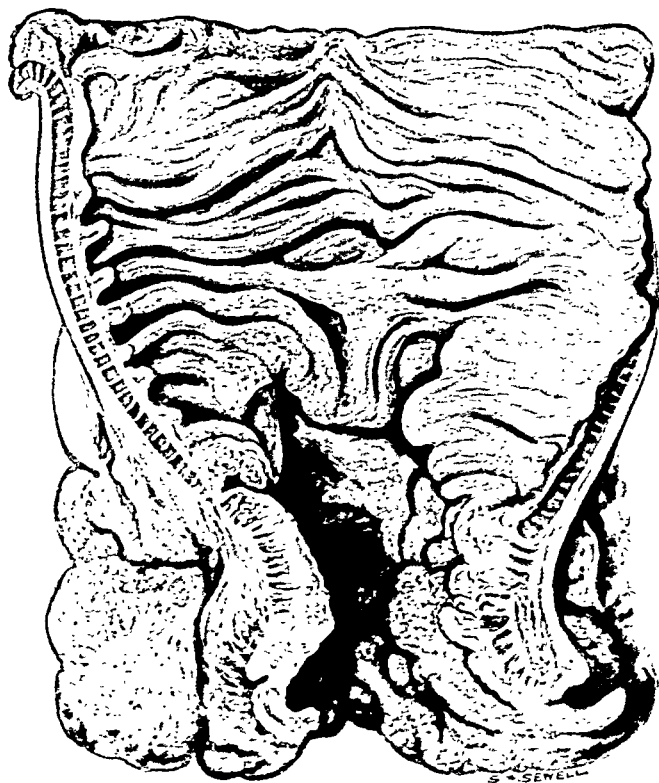
CARCINOMA OF RECTUM.

(PERFORATION.)

A rectum divided longitudinally from behind.

In the centre of the specimen is an annular ulcer varying from $\frac{1}{2}$ to $1\frac{1}{2}$ in. in width. Its edges are raised and in parts everted, its floor is necrotic, and on the anterior surface is a perforation which leads into the recto-vesical pouch of the peritoneum. The peritoneum surrounding the perforation is coated with inflammatory exudate.

Outside the rectal wall, at the level of the growth, the cellular tissue is thickened. No enlarged lymph-glands can be seen.

The rectum is dilated above the stricture, but there is no hypertrophy of its muscular coat. The mucous membrane has lost its rugæ through distension, and shows a group of shallow ulcers near the upper edge of the specimen.

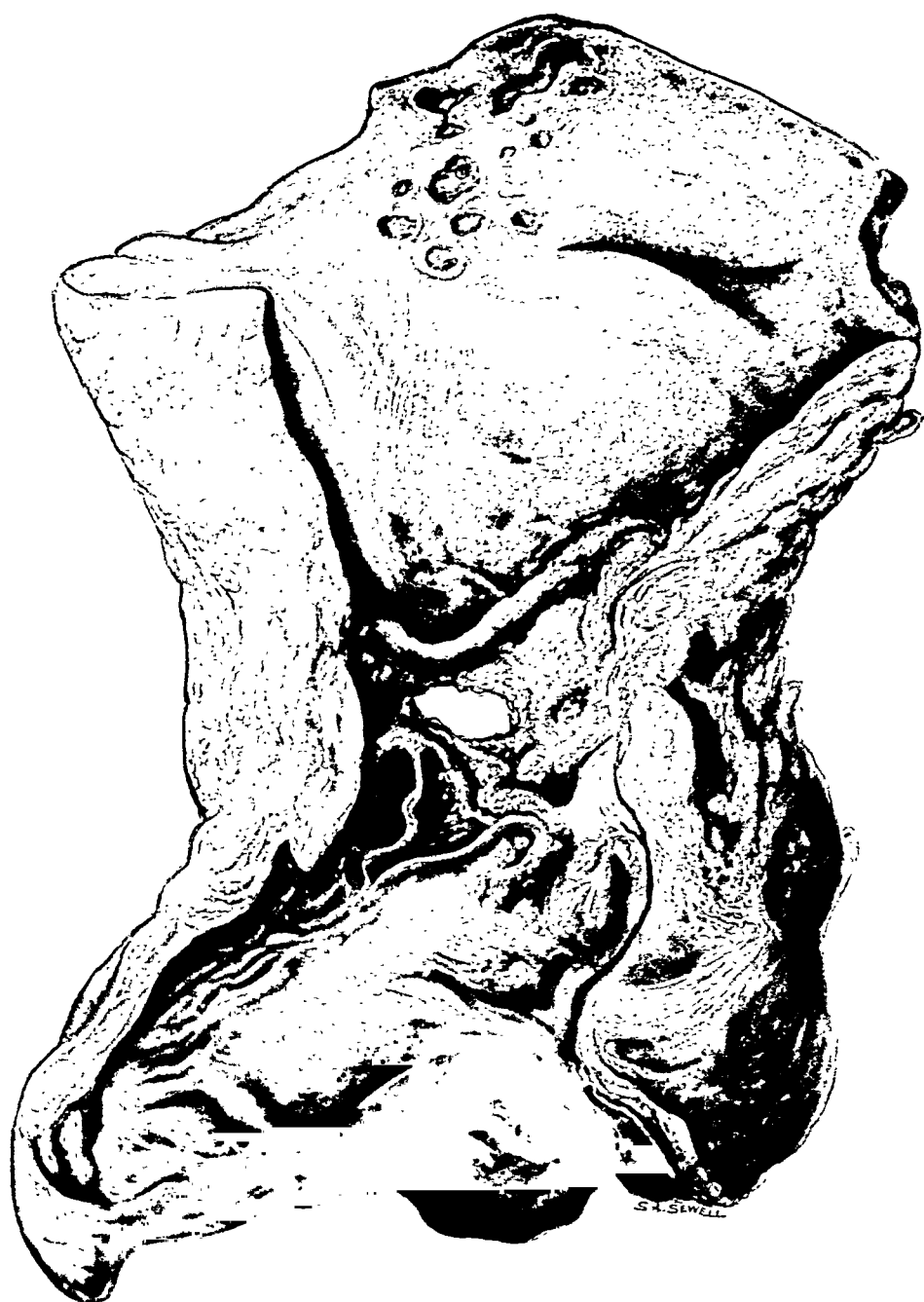*Museum of University College Hospital*, 12.B.D.3

MICROSCOPIC STRUCTURE.—Columnar-cell carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 60, who came to hospital complaining of constipation for five weeks. He was given an enema, which relieved him, and he went home again.

Six days later he came back saying that he had been vomiting ever since his first attendance. He showed signs of peritonitis and was explored immediately. There was turbid fluid in the peritoneal cavity and a perforation in the anterior wall of the rectum. This was closed by suture. The carcinoma was about 6 in. from the anus. He died on the third day after operation.
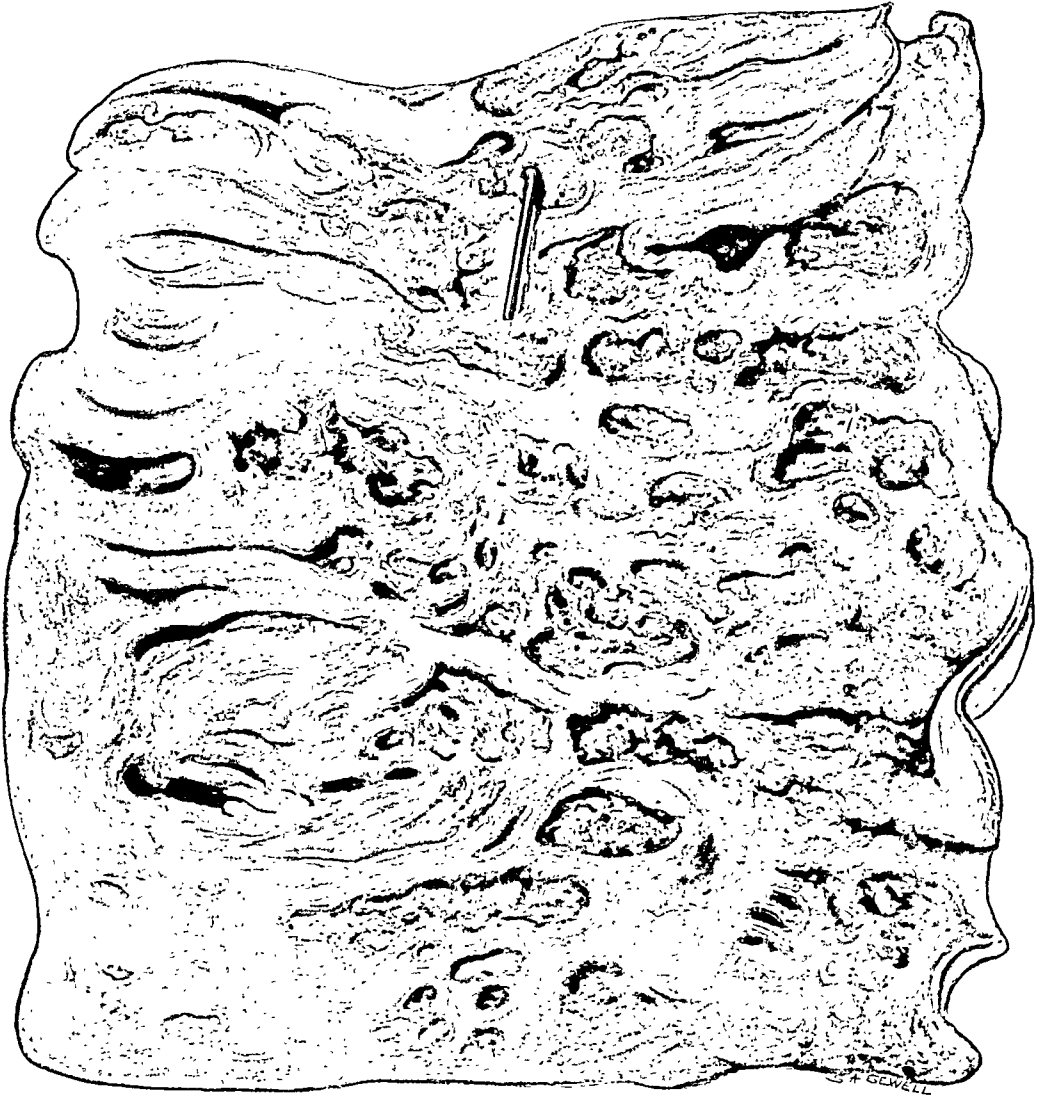
AUTOPSY.—The body was not emaciated. The large intestine was greatly distended and very friable. The mucous membrane contained many ulcers, several of which had perforated into the peritoneal cavity.

CARCINOMA OF RECTUM.

S.A.SEWELL

Perforation of ascending colon.

Part of the ascending colon from the preceding case (U.C.H. 12.B.D.3) divided by longitudinal section.

The mucous membrane is pitted by a large number of irregular ulcers. The rod marks a perforation in the base of one of the ulcers.

Museum of University College Hospital, 12.B.D.4

XLIII. EPITHELIOMA OF THE ANAL CANAL.

SQUAMOUS epithelioma of the anal canal is much less common than carcinoma of the rectum. It arises in the lower third of the anal canal or at the anal margin and may spread either up the anal canal into the rectum or radially from the anal margin out on to the skin covering the ischio-rectal fossa. In the early stages of the disease the degree of infiltration of the base is variable, with a corresponding variation in the induration of the growth on clinical examination. The surface may be ulcerated or warty.

When an epithelioma spreads up the anal canal the clinical appearance of the ulcer may be considerably modified by secondary septic infection, and this factor, together with the consequent irritation of the anal sphincter, largely determines the amount of pain experienced by the patient.

When an epithelioma spreads outwards from the anal margin it tends to grow radially at a greater rate than around the circumference of the anus. Less commonly it takes the form of a linear track which expands in the skin at a short distance from the anal margin. In such cases the element of infection is minimal.

Epithelioma of the anal canal gives rise to secondary deposits in the inguinal lymph-glands. It is a relatively benign and radio-sensitive growth.

XLIV. SARCOMA OF THE RECTUM.

SARCOMA of the rectum is a rare disease. It may be composed of any of the types of cell usually found in similar growths elsewhere.

The tumour first appears in the submucosa. from which it projects into the lumen of the rectum as a nodular mass. This may ulcerate early so as to resemble closely the ordinary carcinomatous ulcer or it may fungate and fill the rectum to the point of obstruction.

Another form sometimes assumed by sarcoma is that of a diffuse infiltration of all coats of the rectal wall and of the extra-rectal cellular tissue. The rectum is then converted into a thick-walled, fixed. and rigid tube. Ulceration of the mucosa is not constant in this form of the disease. Visceral and glandular metastases appear early.

Several specimens of melanotic sarcoma and a few of endothelioma of the rectum occur in pathological museums.

EPITHELIOMA OF ANAL CANAL.

A rectum and anal canal with the surrounding skin divided by longitudinal section.

In the anal canal is a superficial ulcer which has the appearance of an anal fissure. Above it and continuous with its upper border is an irregular ulcer with raised edges and irregular floor, which extends into the rectum for about one inch.

Museum of University College Hospital, 12.B.D.15

MICROSCOPIC STRUCTURE.—Squamous epithelioma.

CLINICAL HISTORY.—The patient was a man, aged 63, who for three weeks had complained of pain on defæcation and of tenderness of the anus on sitting.

On examination there was a tender ulcer in the posterior part of the anal canal. The upper part of this ulcer had hard everted edges and was continued into the rectum. The right inguinal lymph-glands were enlarged.

Colostomy was followed four weeks later by perineal excision of the rectum.

No after-history.

SARCOMA OF RECTUM.

The lower part of a rectum with the anus opened by longitudinal incision.

A short distance above the anus there is a malignant ulcer nearly 2 in. in diameter. The edge is slightly raised and everted and the floor is deeply excavated. The perirectal connective tissue is infiltrated by growth. The mucous membrane of the rectum is otherwise normal.

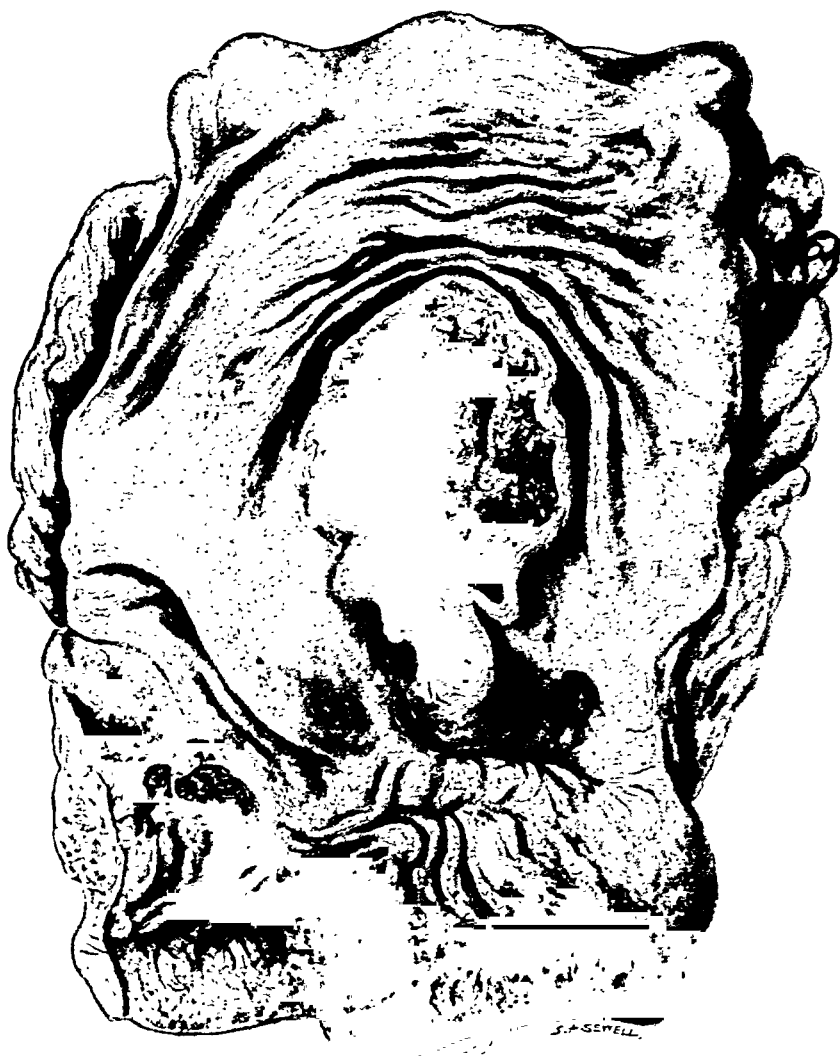*Museum of the College of Medicine, University of Durham. Newcastle-upon-Tyne, 373/20*

MICROSCOPIC STRUCTURE.—Spindle-cell sarcoma.

CLINICAL HISTORY.—The patient was a man, aged 61, who came to hospital complaining of difficulty with micturition and of a discharge from the rectum. He had had retention of urine, relieved by instruments, on a previous occasion. For nine weeks he had suffered pain after food and on defæcation. A brown and slimy discharge came from the anus independently of defæcation. He always had a feeling of not having emptied the bowel properly and was unable to pass a solid motion although he went to stool about thirty times a day.

At operation colostomy was performed, and three weeks later the rectum was excised by the perineal route. The patient died a few days after the second operation.

AUTOPSY.—There were two small secondary growths in the lungs, but none in the liver.

SARCOMA OF RECTUM.

Atlas of Pathological Anatomy

MELANOTIC SARCOMA OF RECTUM.

The lower half of a rectum together with a small part of the posterior vaginal wall. The rectum has been opened by vertical incision from the front.

A lobulated, pigmented, fungating growth is situated immediately above the sphincter. It springs from the anterior and left lateral wall of the bowel and has invaded the cellular tissue between the rectum and vagina, the wall of which is thickened but not ulcerated. Above the cut edge of the vagina a plaque of growth is present in the perirectal tissues, and several glands enlarged by growth have been dissected on the outer surface of the bowel. The rectum is considerably dilated above the growth but is not ulcerated or inflamed.

Hunterian Museum, R.C.S., 1690.2

MICROSCOPIC STRUCTURE.—Spindle-celled melanotic sarcoma. In a section from an enlarged gland the growth was not pigmented and the cells show an alveolar arrangement.

CLINICAL HISTORY.—The patient was a married woman, aged 65, who had had a mucous discharge tinged with blood from the rectum fifteen months before admission to hospital. During the last six months the bleeding increased, and for three weeks she had had pain lasting several hours after defæcation.

MELANOTIC SARCOMA OF RECTUM.

S.H.SEWELL

Atlas of Pathological Anatomy

ENDOTHELIOMA OF RECTUM.

A rectum opened by longitudinal section.

The bowel is completely encircled by a growth which is ulcerated over most of its surface. The muscular coat is thickened and infiltrated by the tumour. The lumen of the rectum is slightly diminished.

Museum of St. Mark's Hospital, 2864

MICROSCOPIC STRUCTURE.—The mucous, submucous, and muscular coats are infiltrated by a tumour which is ulcerated over parts of its surface. The tumour is composed of spheroidal and polygonal cells with large nuclei. Many of these cells are arranged in groups around primitive vessels. The growth has the general histology of an endothelioma arising from vascular endothelium. It has spread into the extrarectal tissues, but the regional lymph-glands do not contain metastases.

CLINICAL HISTORY.—The patient was a young woman who was at first thought to have either a tuberculous or malignant ulcer constricting the rectum.

A portion of growth was removed through the sigmoidoscope and was found to have the structure of an endothelioma. The rectum was removed by perineal excision, from which the patient made a good recovery.

ENDOTHELIOMA OF RECTUM.

A. K. MAXWELL.
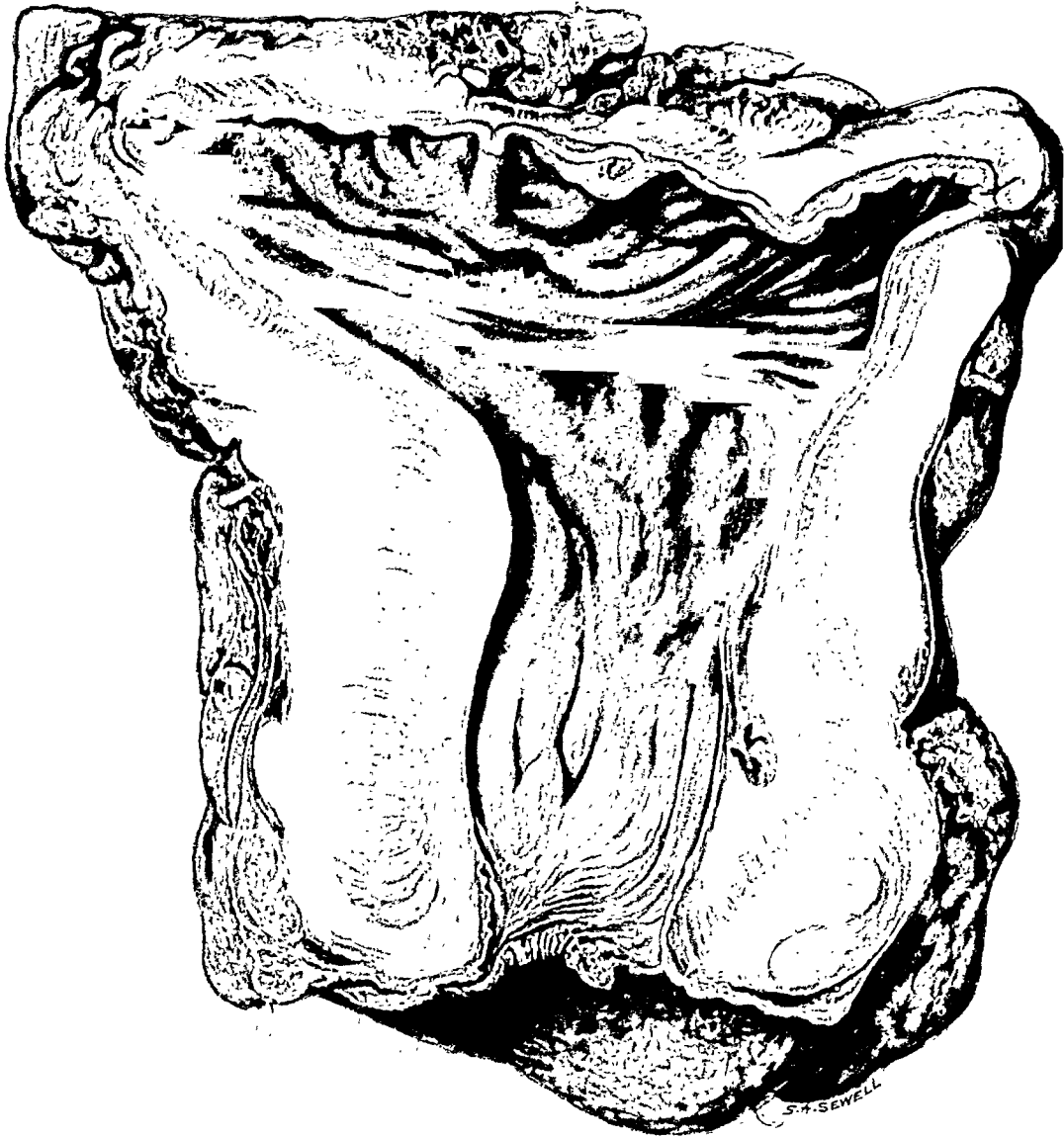
An anal canal with the adjoining part of the rectum divided by longitudinal section.

The walls of the anal canal and lower rectum are infiltrated and greatly thickened by a white growth in which the sphincter can still be traced faintly. The surface of the mucous membrane is intact. On the reverse side of the specimen the growth extends out into the fat of the ischiorectal fossa and perirectal tissues.

Hunterian Museum, R.C.S., 1562.3

MICROSCOPIC STRUCTURE.—Round-celled sarcoma.

CLINICAL HISTORY.—The patient, who was aged 68, had suffered from obstinate constipation and swelling round the anus for two months. After a preliminary colostomy the specimen shown was removed by the perineal route. Death from shock.

XLV. CARCINOMA OF THE COLON.

CARCINOMA is a common disease of the colon. Its subjects are usually over the age of 40 and show a slight preponderance of males. The colon in which a carcinoma arises may have been healthy beforehand. but often it contains simple adenomata, sessile or pedunculated, and occasionally a carcinoma has been found in association with diverticula. The relatively rare condition known as 'fibromatosis' or 'polyposis' of the colon is often a precursor of malignant disease. Carcinoma of the colon commences in the glands of the mucous membrane and is composed of columnar cells lying in a fibrous stroma.

The naked-eye appearance of the growth depends on whether it spreads chiefly by infiltration of the wall or by fungation into the lumen of the bowel. The usual form is that of the annular stricture, and its characteristic site is the pelvic colon. It is formed by a narrow band of growth which extends round the circumference of the intestine in the lymphatics of the submucosa until a complete ring is made. The fibrous stroma of the carcinoma contracts as the epithelial cells advance so that the lumen of the gut is concentrically diminished until a point is reached at which obstruction occurs. The rate of progress varies. but is. on the average, such that about a year elapses between the beginning of the growth and the onset of clinically recognizable symptoms of obstruction. From the peritoneal aspect the appearance is that which would be produced by tying a string round the bowel. On the mucous surface the growth is raised and ulcerated. and on account of the eversion of its edges is longer than it appears on the outside. Occasionally a more cellular type of carcinoma will extend longitudinally as well as around the gut. so that the affected segment of intestine is replaced by a tube of growth.

The fungating type of carcinoma of the colon is seen in its most character-istic form in the cæcum and ascending colon. where it makes a bulky tumour encircling the intestine for a distance of 2 or 3 in. Owing to constriction of its blood-supply by infiltration of the base and infection of its surface from the contents of the bowel. ulceration is extensive and may maintain the patency of the lumen enough to prevent the appearance of obstructive symptoms even in an advanced stage of metastasis. A carcinoma of the colon which grows towards the lumen of the bowel may assume a polypoid form and become the starting-point of an intussusception. From the peritoneal aspect the attachment of this kind of tumour is often marked by a pucker or by a local invagination of the wall. On the mucous surface. if the tumour is composed chiefly of epithelium. there is usually some ulceration round its neck. If the tumour is composed mainly of infiltrated submucosa. the mucous membrane covering it may appear normal to the naked eye. It rarely happens that a carcinoma of the colon takes the form of a circular ulcer with raised. everted edges as in the common variety of malignant disease of the rectum.

Colloid (mucinous) degeneration is not unusual in carcinoma of the colon. It converts the growth into a bulky tumour with a gelatinous appearance on

K

section, but has no influence on the degree of malignancy. In all ulcerating carcinomas of the colon, especially those of the more bulky kind, inflammatory changes consequent to the ulceration add to the size of the tumour and often contribute to its fixation to surrounding structures.

In the wall of the colon carcinoma spreads chiefly in the submucosa, and, as described above, at right angles to, rather than in, the long axis of the gut. Occasionally a second carcinoma, or even more than one, is found at a considerable distance below the original growth. The path taken by the epithelial cells which form the metastatic deposit is probably along lymphatic channels, either in the submucosa or outside the intestine altogether. A secondary growth arising in this way may be larger and more easily recognized clinically than the primary from which it sprang.

Direct spread of carcinoma beyond the wall of the colon involves the retroperitoneal cellular tissue, the peritoneum, or any viscus, such as the bladder or a coil of small intestine, to which the growth may have adhered. Infection following the track of the carcinoma may then cause a pericolic abscess, peritonitis, or a fistula. Where the balance between ulceration and stenosis is so precisely adjusted as to preclude obstruction, perforation is often the first event to drive the patient to the notice of a surgeon.

Extension along lymphatic channels in its earlier stages is most clearly demonstrated and its extent is most readily determined where infection is minimal. Lymphatic vessels distended with white growth can often be seen beneath the peritoneal coat of the bowel in the immediate neighbourhood of the carcinoma. The first glands to be invaded are those which lie close to the colon, and from these there is a progressive spread through the glands on the colic arteries to those around the superior and inferior mesenteric vessels. The latter group may be enlarged by growth without apparent involvement of the intermediate stations. Estimation of the extent of glandular invasion at operation is apt to be stultified by coincident inflammatory enlargement.

Secondary growths in the liver and peritoneal cavity appear relatively late. Metastasis in the lungs and in the skeleton is very rare.

The natural ending of a carcinoma of the colon is in obstruction of the bowel. The development of obstruction is accompanied by compensatory hypertrophy of the muscular coat of the gut on the proximal side of the growth, and its later stages are marked by distension and by a great increase in the infectivity of the contents of the bowel above the stricture. Ulceration of the mucous membrane may end in perforation of the colon just above the growth or of the cæcum.

CARCINOMA OF TRANSVERSE COLON.

Part of a transverse colon divided by longitudinal section.

In the middle of the specimen is a stricture caused by an annular growth ¾ in. in length at the level of section. The growth is white in colour with raised edges and ulcerated surfaces. The lumen is reduced to an irregular slit. The muscular coat of the bowel is drawn up into the edges and is destroyed opposite the centre of the carcinoma. The bowel on the proximal side of the stricture is dilated and injected and its muscular coat is hypertrophied. The growth extends into it in the form of an oval ulcer about ¾ in. in its long diameter. The bowel on the distal side is normal, though it has been stretched by being stuffed with cotton-wool during the process of preservation. The surface of the colon is marked by a deep constriction at the site of the carcinoma. There are no adhesions to surrounding parts.

Hunterian Museum, R.C.S., 6543.1

MICROSCOPIC STRUCTURE.—Columnar-celled carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 57, who had suffered from intestinal obstruction for four days with frequent vomiting, abdominal distension, and collapse. Cæcostomy was followed by resection of the parts shown ten days later, and the cæcostomy was closed in another ten days.
    Recovery. No after-history.
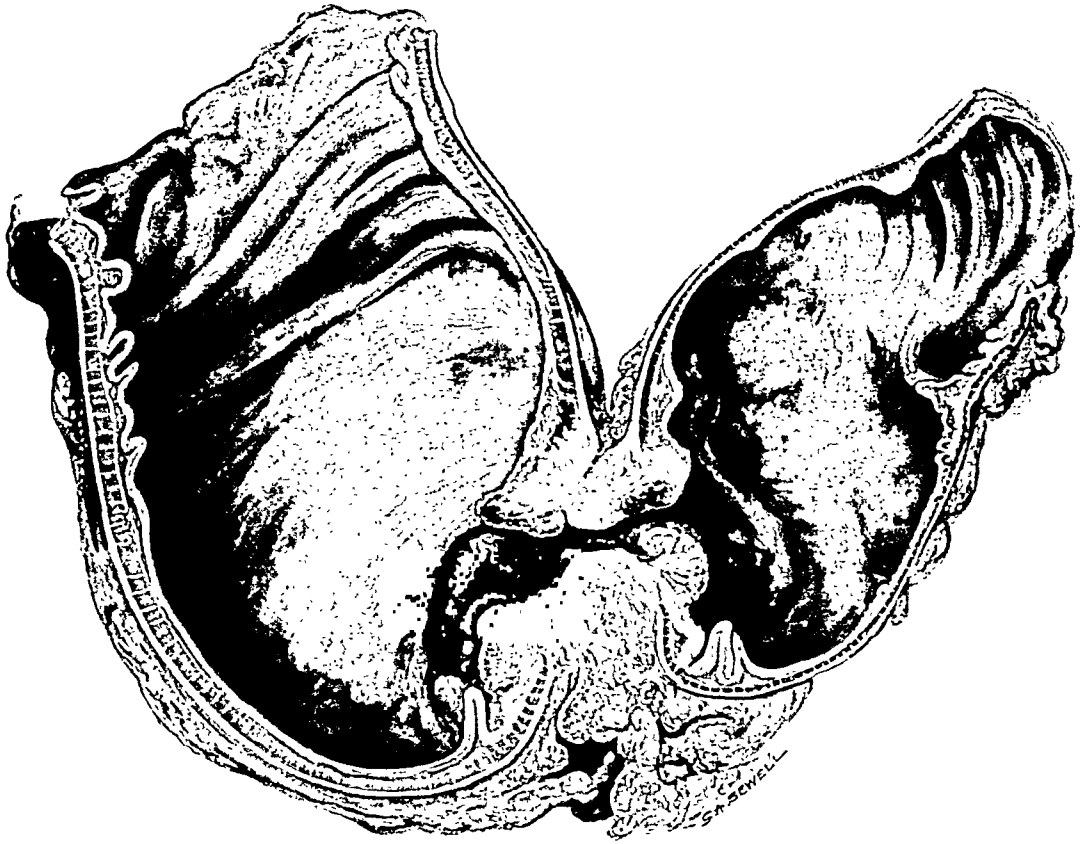
SECTION.

HUNTERIAN MUSEUM, R.C.S. 6543.1

CARCINOMA OF TRANSVERSE COLON.

EXTERNAL SURFACE.

NO. 31—SUPPLEMENT

K 1

CARCINOMA OF PELVIC COLON.

Part of a pelvic colon.

The colon is tightly constricted by an annular carcinoma. On the proximal side of the stricture the gut is dilated and hypertrophied.
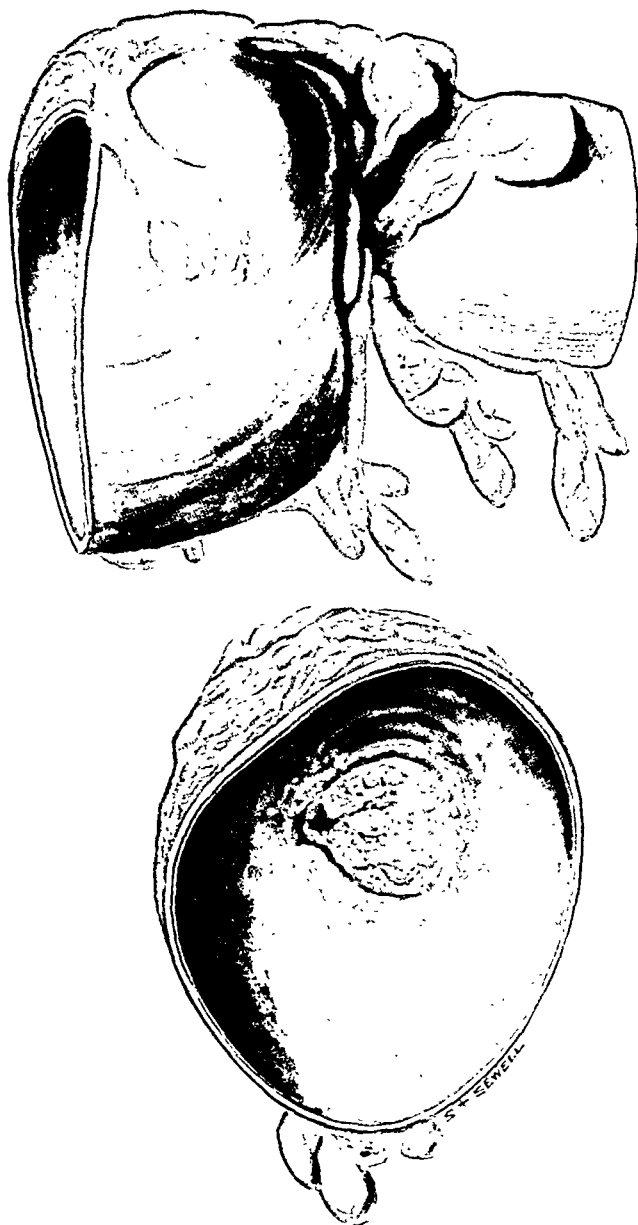
Hunterian Museum, R.C.S., 1980.1

MICROSCOPIC STRUCTURE.—Columnar-celled carcinoma.

CLINICAL HISTORY.—The patient was a woman, aged 63, who was admitted to hospital suffering from chronic intestinal obstruction. The abdomen was explored but the growth was not discovered. She died of congestion of the lungs.

AUTOPSY.—No secondary growths found.

CARCINOMA OF TRANSVERSE COLON.

Part of a transverse colon with the attached omentum divided by longitudinal section.

In the middle of the specimen is a malignant stricture 1½ in. long. The growth has largely replaced the normal coats of the bowel and is invading the fat at its upper margin. The edges of the carcinoma are smooth, raised, and covered by mucous membrane. The centre is superficially ulcerated. The intestine on the proximal side of the stricture is moderately dilated but not hypertrophied. The mucous lining is not ulcerated.

Hunterian Museum, R.C.S., 6544.1

MICROSCOPIC STRUCTURE.—Columnar-celled carcinoma.

CLINICAL HISTORY.—The patient was a fat woman, aged 58, who had had intestinal obstruction for four days. The onset was sudden with colicky pain, vomiting, and absolute constipation. On examination the abdomen was slightly distended and tender on palpation. No tumour was felt. At operation the growth was near the splenic flexure, and the cæcum and ascending and transverse colon were greatly distended. There was no enlargement of the regional lymph-glands. Cæcostomy was followed by resection of the specimen shown. The cæcostomy was closed in another two weeks.

No after-history.

S. A. SEWELL

HUNTERIAN MUSEUM, R.C.S., 6544.1

CARCINOMA OF PELVIC COLON.

Part of a pelvic colon divided by longitudinal section.

A solid white growth encircles the middle of the specimen and reduces the lumen to a mere slit. The carcinoma has apparently commenced on the concave side of the loop of bowel, where it extends for a considerable distance on either side in the submucosa, and at several points has perforated the muscle and is invading the fat of the mesocolon. It is less extensive on the opposite side of the gut. On the reverse side in the specimen are several enlarged glands in the mesocolon.

Hunterian Museum, R.C.S., 1728.1

MICROSCOPIC STRUCTURE.—Spheroidal-celled carcinoma. The growth can be traced to the glands of the mucosa, but the cells have lost their columnar shape.

CLINICAL HISTORY.—The patient was a woman who suffered from increasing intestinal obstruction for fourteen days. Before admission to hospital the obstruction had become absolute. A colostomy was followed after two weeks by excision of the tumour and end-to-end union of the gut.

AFTER-HISTORY.—The patient was well one year later.

S.A.SEWELL

HUNTERIAN MUSEUM. R.C.S., 1728.1

CARCINOMA OF PELVIC COLON.

Part of a pelvic colon opened by longitudinal section.

In the lower part of the specimen is an ulcerated carcinoma which involves about half the circumference of the bowel. The muscular coat of the colon is hypertrophied and there is an enlarged gland in the mesocolic attachment. By the side of the specimen is a pedunculated papilloma, which was attached to the bowel a short distance below the malignant growth.

Museum of the Cancer Hospital, London, 461.

MICROSCOPIC STRUCTURE.—Columnar-celled adeno-carcinoma infiltrated by inflammatory cells.

CLINICAL HISTORY.—The patient was a woman, aged 65, who had complained of increasing pain and hæmorrhage on defæcation for six months. On examination a large mass, which was not fixed, was felt in the rectum. reaching nearly to the anus.

At operation there was an intussusception at the apex of which was a simple papilloma with a carcinoma a short distance above it. Resection of the affected segment of gut with end-to-end anastomosis was followed by a good recovery, and the patient left hospital free from all symptoms.

No after-history.

Atlas of Pathological ...
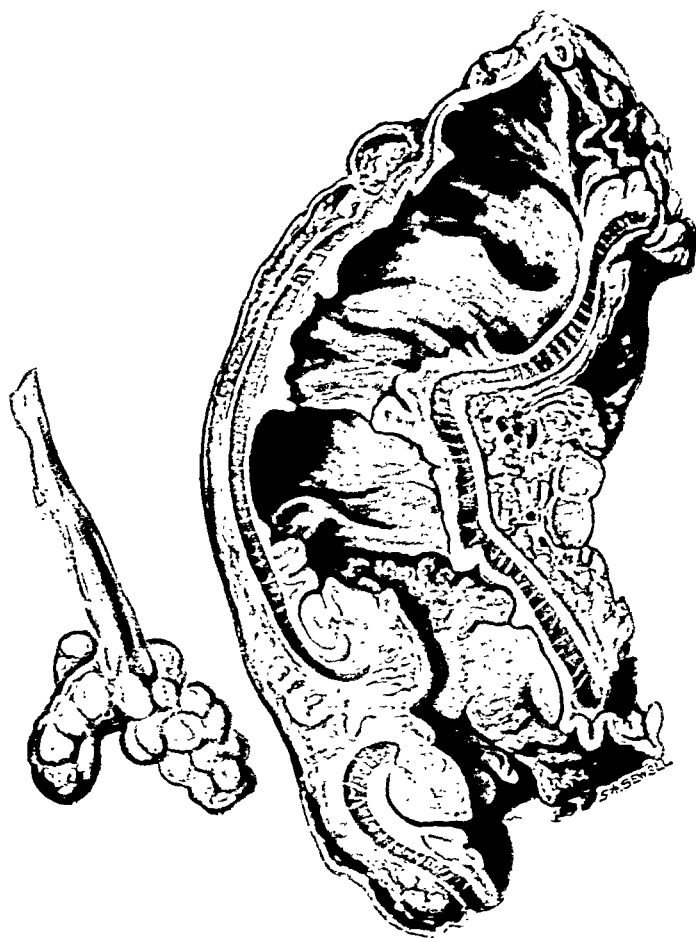
CARCINOMA OF CÆCUM.

A cæcum with the adjacent parts of the ileum and ascending colon. The anterior wall of the cæcum and colon has been removed.

A fungating tumour completely encircles the ileo-cæcal junction and lower portion of the cæcum. The lymph-nodes in the neighbouring part of the mesentery were palpable.
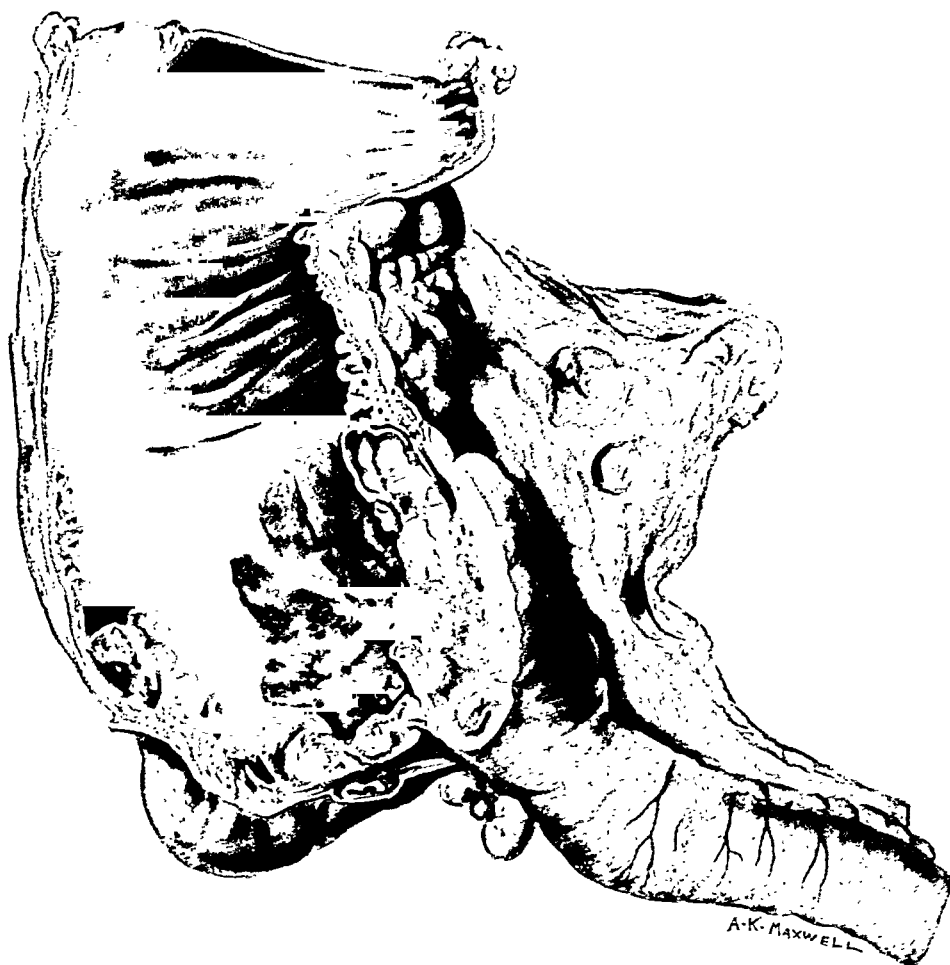
Museum of St. Mark's Hospital, 2597.

MICROSCOPIC STRUCTURE.—The tumour is an adeno-carcinoma which shows colloid degeneration in parts. The growth has spread by direct continuity through the wall of the cæcum. The regional lymph-glands are enlarged as the result of inflammatory reaction but contain no metastases.

The parts shown were removed by operation and the patient made an uninterrupted recovery.

AFTER-HISTORY.—Six months later the patient was in good health.

CARCINOMA OF CÆCUM.

A·K·MAXWELL

CARCINOMA OF CÆCUM.

A cæcum with parts of the ascending colon and ileum opened by longitudinal section.

The greater part of the cæcum and lower portion of the ascending colon have been involved in the growth of a tumour which has replaced their walls, with the exception of the peritoneal coat. The lumen is considerably diminished and the inner surface of the growth is necrotic. The ileo-cæcal valve has been invaded.

Museum of the Cancer Hospital, London, 1112.

MICROSCOPICAL STRUCTURE. — Columnar-celled carcinoma with colloid degeneration.

CLINICAL HISTORY.—The patient was a woman, aged 62, who had had shooting pains in the right iliac fossa for five months. Her appetite was good and the bowels were regular. The tumour was discovered by her doctor and had not been noticed by the patient herself.

The specimen shown was removed by operation and an end-to-end ileo-transverse anastomosis made. She died on the eighth day after operation from paralytic ileus.

CARCINOMA OF CÆCUM.

S. & SEWELL

Atlas of Pathological Anatomy

CARCINOMA OF TRANSVERSE COLON.

Part of a transverse colon, divided at right angles to its long axis.

The lower specimen shows the distal surface of a polypoid growth which projects into the lumen of the bowel from the mesocolic border. The tumour is nodular and its surface is covered by intact mucous membrane.

The upper specimen is a section from the middle of the growth. It shows that the carcinoma is situated in the submucosa from which it has begun to invade the muscle. The muscular wall of the bowel above the growth is hypertrophied.

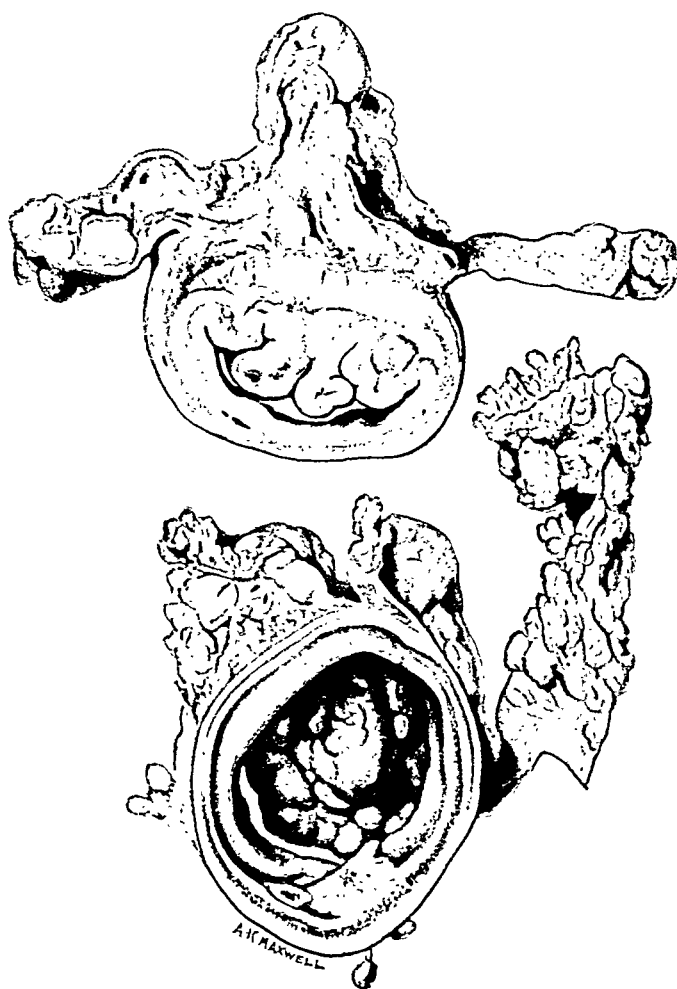*Museum of University College Hospital.* 11Y1

MICROSCOPIC STRUCTURE.—Columnar-celled carcinoma.

CLINICAL HISTORY.—The patient was a man, aged 43, who for seven months had had intermittent pain in the left hypochondrium, with constipation, vomiting, and the passage of blood. Between the attacks he felt quite well. When seen during an attack of pain there was a hard tender mass in the left iliac fossa. It was thought to be an intussusception. Between attacks the lump disappeared.

At operation a small hard tumour was found in the middle of the transverse colon. Below this, the large intestine was normal. A portion of the transverse colon containing the growth was removed and the ends were united by suture. A temporary cæcostomy was established. The patient left the hospital in good health.

No after-history.

A.K.MAXWELL

CARCINOMA OF PELVIC COLON.

A pelvic colon, at one end of which a circular opening has been cut.

The colon is greatly distended. Its wall is hypertrophied and the peritoneum covering it is injected. A few flakes of recent lymph are attached to the surface.

A growth, having the form of an annular stricture, can be seen through the opening in the upper part of the specimen. At the back, the bowel distal to the growth is of normal size.
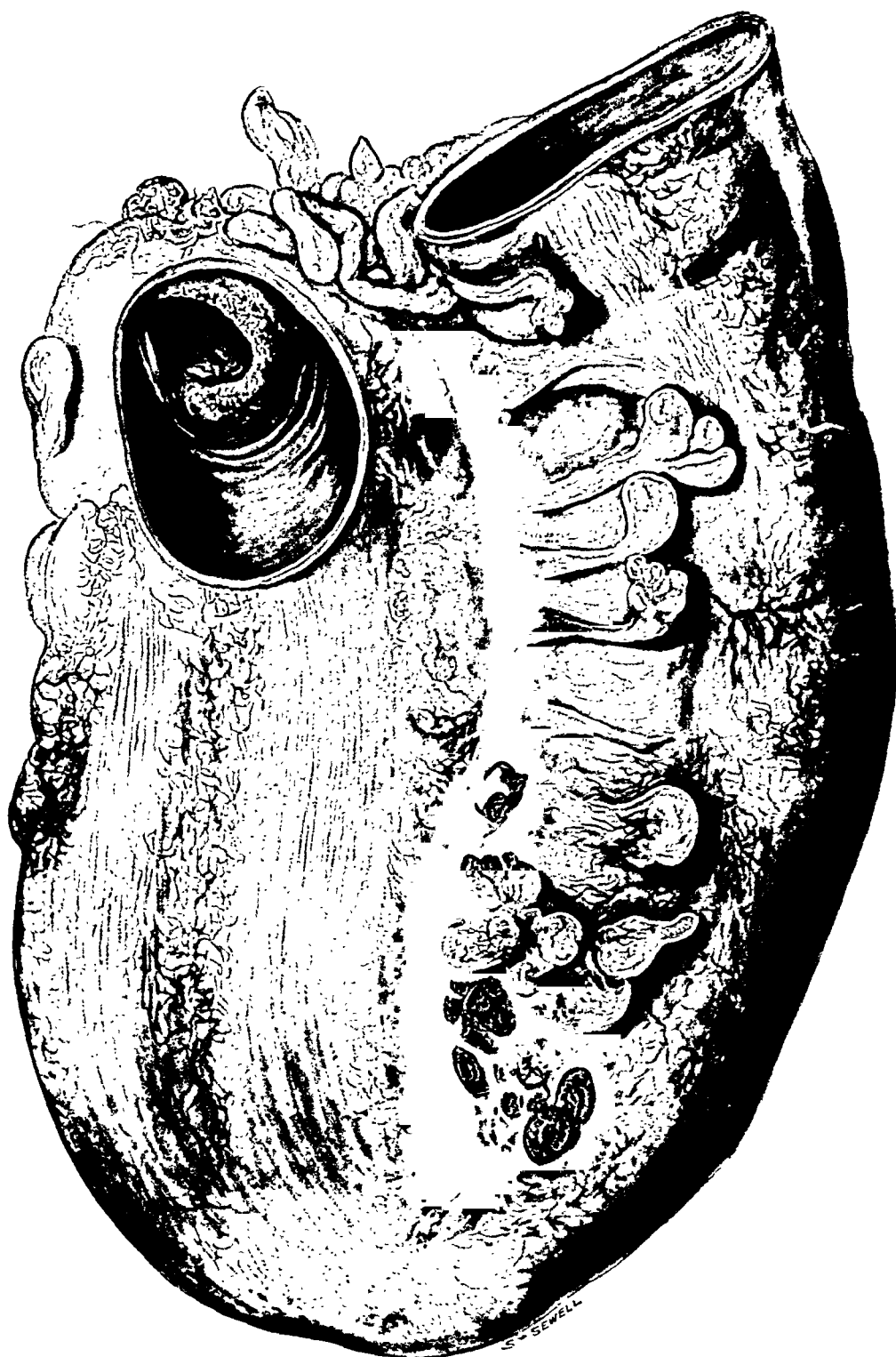
Museum of St. Bartholomew's Hospital, L.203

MICROSCOPIC STRUCTURE.—Columnar-cell carcinoma.

CLINICAL HISTORY.—The patient was a male deaf-mute, aged 79, who had had symptoms of obstruction of the bowel for three days. There was fæcal vomiting. He died on the fifth day from peritonitis and shock.

AUTOPSY.—Perforation of the cæcum.

S. S. SEWELL

CARCINOMA OF PELVIC COLON.

A portion of a pelvic colon divided by longitudinal section.

For a length of 4 in. the mucous membrane and submucosa are thickened by carcinomatous infiltration. The muscular coat is hypertrophied. The growth has extended out into the mesocolon.
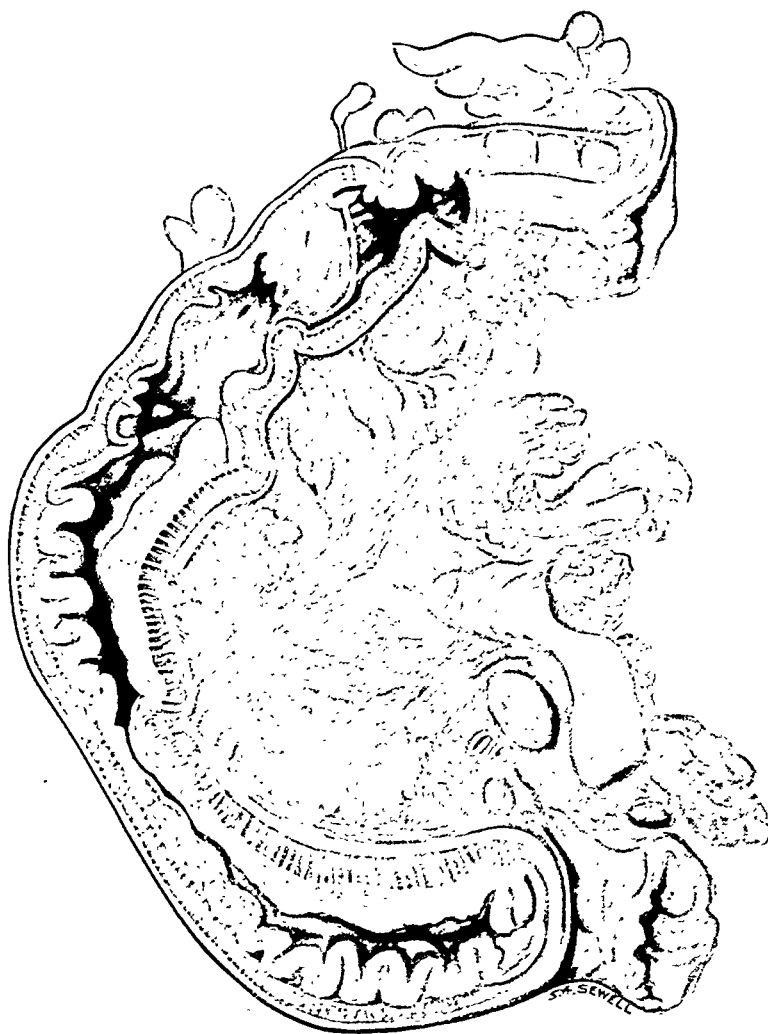
Hunterian Museum, R.C.S., 1760.1

MICROSCOPICAL STRUCTURE.—Atypical columnar-celled carcinoma. The cells are mostly spheroidal and are arranged in narrow lines which at some points are continuous with the crypts of Lieberkühn. Here and there in the muscle are some imperfectly formed tubules, lined by columnar cells and with a sharply defined lumen.

CLINICAL HISTORY.—The patient was a woman aged 34. Four months before admission she noticed a lump on the left side and pain on the right side of the abdomen, which gradually increased in size. There was no constipation. Food either passed rapidly through her, or was vomited.

On examination there was a movable tumour and free fluid in the abdomen. A second tumour could be felt behind the uterus. At operation two ovarian tumours were removed and three or four pints of peritoneal fluid. The tumour of the pelvic colon was then found and removed. The ovarian tumours were secondary carcinoma.

The patient died six months later.

CARCINOMA OF PELVIC COLON.

Atlas of Pathological Anat.

XLVI. SARCOMA OF THE INTESTINE.

SARCOMA is a rare disease of the intestine, but occurs at all ages and more often in the small than in the large bowel. The predominant cellular type is a lympho-sarcoma, which commences in the lymphoid tissue of the mucous membrane and infiltrates all coats. The intestine is converted into a rigid, thick-walled tube without serious diminution of the lumen in the earlier stages. The mesentery of the affected loop is involved by secondary deposits in the lymph-glands and by a direct invasion of its cellular tissue by the tumour. A mass of considerable size is formed in this way and may be recognizable clinically by the time obstruction commences.

In other cases the growth begins in the submucosa and projects into the lumen of the gut as a polypoid tumour. It may have a round-cell, spindle-cell, or fibro-sarcomatous structure, and may first attract attention by causing an intussusception. If the base of attachment is broad and encircles the gut the growth fungates into the lumen and produces a gradually increasing obstruction.

The least common form of sarcoma of the intestine is that which arises in the subserous layer and projects from the peritoneal surface. Obstruction may be caused either by pressure of the tumour from the outside or by infiltration at its base.

Many cases are recorded in which the local removal of a sarcoma of the intestine has been followed by a long period of freedom from symptoms, and it appears probable that many such tumours are of a relatively benign type.

SARCOMA OF SMALL INTESTINE.
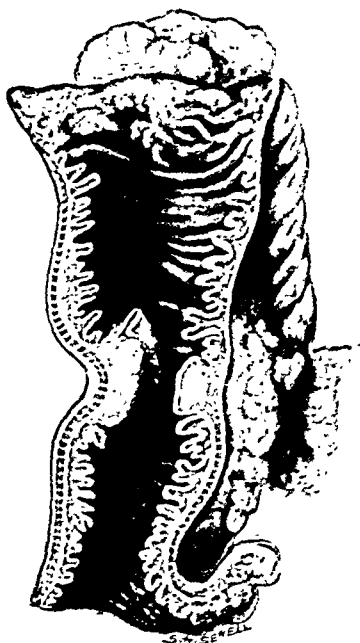
Three inches of small intestine divided by longitudinal section.

In the centre of the specimen the lumen is reduced by a yellow growth half an inch long which has replaced the mucous and submucous coats. The tumour has extended through the muscle and has infiltrated the peritoneum. The mesenteric half of the circumference of the gut is not involved.

A mesenteric lymph-gland enlarged by growth is shown.

Hunterian Museum, R.C.S., 6534.1

MICROSCOPIC STRUCTURE.—The tumour is composed of slender interlacing columns of spheroidal cells separated by a scanty fibrous stroma. The columns of cells penetrate the muscular coat into the serosa and extend between the glands of the mucous membrane without having any direct connection with them.

CLINICAL HISTORY.—The patient was a man, aged 60, who was operated upon for irreducible inguinal hernia with symptoms of obstruction. The portion of small intestine containing the growth lay within the sac and was excised.

AFTER-HISTORY.—Well six months later.

SARCOMA OF ILEUM.

A portion of small intestine divided by longitudinal section.

A polypoid tumour is attached by a narrow stalk to the wall of the bowel and has produced a deep indentation on the peritoneal surface.

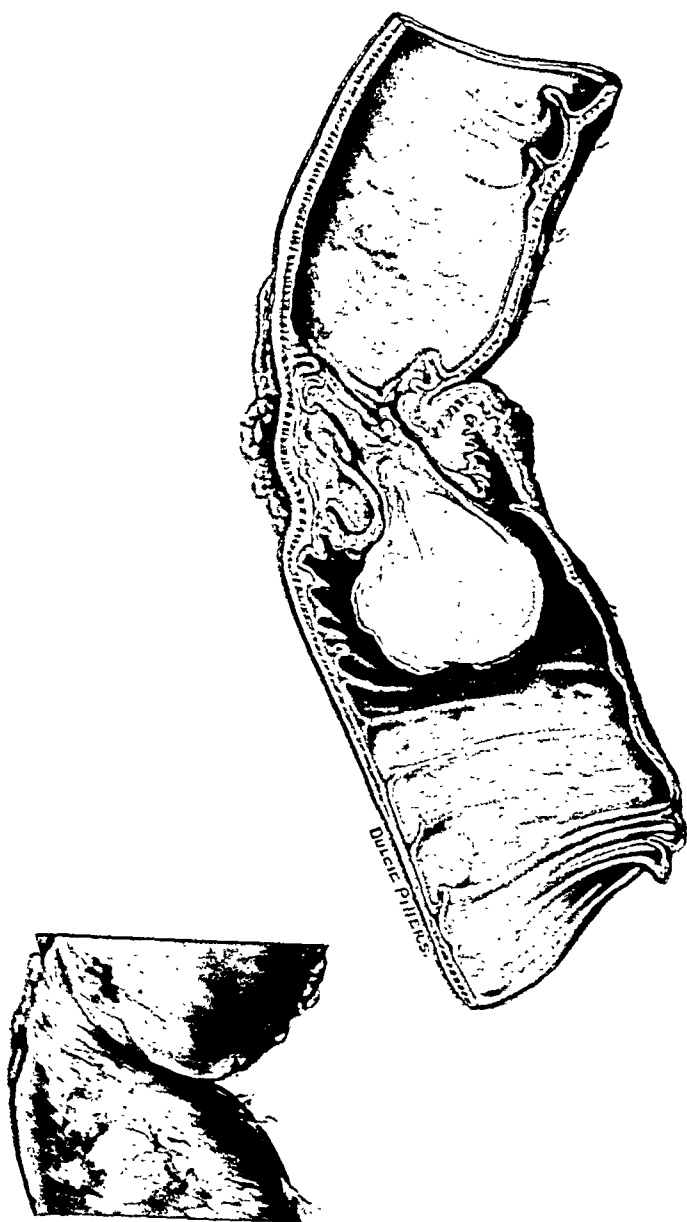*Museum of the University of Bristol.*

MICROSCOPIC STRUCTURE.—The tumour is a fibro-sarcoma arising in the submucosa and perforating the mucous membrane at several points. The muscular wall of the intestine is not deeply involved.

CLINICAL HISTORY.—The patient was a woman, aged 52, who for four months had complained of griping pains in the lower abdomen, often accompanied by vomiting.

At operation a growth was found 2 ft. above the ileocæcal valve. It was œdematous and covered with lymph. The bowel above the tumour was much dilated. The growth was excised with 4 in. of intestine above and below, and an end-to-end suture performed.

AFTER-HISTORY.—The patient is alive and well seven years after the operation.

SARCOMA OF ILEUM.

Dulcie Pillers

SARCOMA OF SMALL INTESTINE.

Part of a small intestine.

For a length of 8 in. the walls of the intestine have been made thick and rigid by a diffuse, opaque, white growth. The lumen is not reduced. On the right side of the specimen a portion of the anterior wall of the intestine has been removed to show the ulceration of its inner surface. The mesentery is invaded by growth.
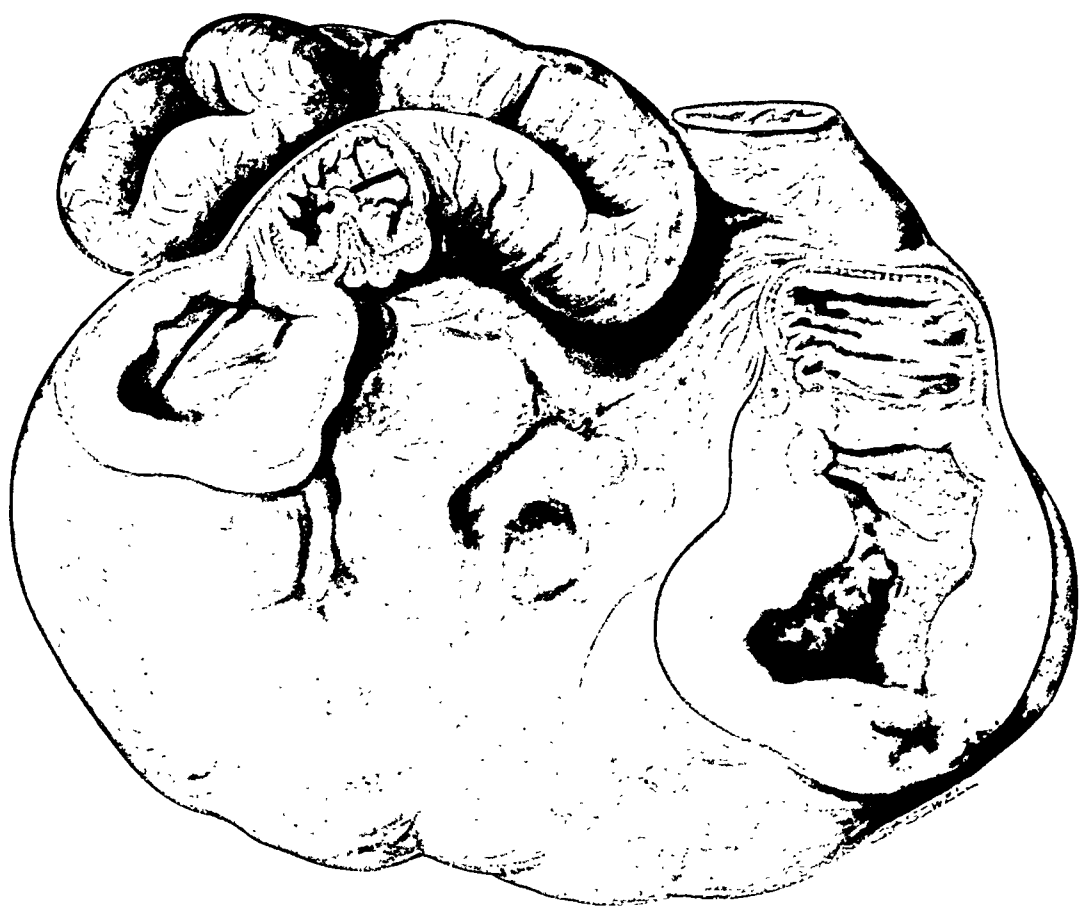
Hunterian Museum, R.C.S., 1562.1

MICROSCOPIC STRUCTURE.—Round-celled sarcoma.

CLINICAL HISTORY.—The patient was a boy, aged 14, who had complained of pain in the right side of the abdomen for two months. He had become pale and thin, but had had no difficulty in retaining food until one week before admission to hospital, when he began to vomit. His bowels were regular.

On examination a hard round swelling extended from the pubes to $1\frac{1}{2}$ in. above the umbilicus. It was fixed, solid, and resonant, and could be felt in the pelvis. The mass was tender on the right side.

At operation the specimen shown was removed together with a second small growth in the intestine some distance away. The tumour recurred locally one month later and the patient was treated by deep X rays and Coley's fluid. He suffered no pain and his bowels remained regular. He died four months after the operation.

FIBRO-SARCOMA OF JEJUNUM WITH ASSOCIATED CYST.

A segment of jejunum with part of an attached cyst.

A smooth hemispherical tumour projects into the lumen of the jejunum on one side, and on its opposite side projects into the interior of the cyst. The cyst is free from the small intestine, to which it is attached only by means of the growth. The large globular cyst, of which only a quarter is preserved, contained blood-clot in the fresh state.

Museum of the Cancer Hospital, London, 991

MICROSCOPIC STRUCTURE.—Fibro-sarcoma richly supplied with blood sinuses and vessels.

CLINICAL HISTORY.—The patient was a woman, aged 35, who had suffered from severe attacks of indigestion with vomiting for three months.

On examination a large cystic tumour could be felt on the left side of the abdomen.

At operation the cyst was attached to the wall of the upper jejunum by a hard mass; 6 in. of jejunum were resected together with the cyst, and the continuity of the gut was restored by side-to-side anastomosis.

AFTER-HISTORY.—There was complete freedom from symptoms for five and a half years. Several attacks of severe colicky pain in the epigastrium for a period of four weeks then led to the patient's re-admission to hospital. It was thought that the growth had recurred, and the abdomen was re-opened. The mesenteric and aortic glands were enlarged and hard, but on microscopic examination showed no sign of growth. The blind ends of the anastomosed bowel were each about 3 in. long and had increased considerably in size. It was concluded that the recent abdominal symptoms were due to accumulation of intestinal contents in these blind ends.

XLVII. SIMPLE TUMOURS OF THE INTESTINE.

SIMPLE tumours of the intestine, apart from the rectum, are rare. Those of connective-tissue origin—fibroma, lipoma, and fibro-lipoma—arise in the submucosa and tend to become polypoid. They then project into the lumen of the bowel and may become the starting-point of an intussusception. Pedunculated subperitoneal tumours, such as fibro-myoma and lipoma, are still less common.

Adenoma and papilloma may occur as isolated examples, but in their most characteristic form tend to be multiple and to be scattered throughout the whole length of both small and large intestine. They arise from the mucous membrane and form pedunculated tumours which project into the lumen of the bowel. They become more numerous in the pelvic colon and usually extend into the rectum. In the lower parts of the large intestine they are apt to ulcerate and to be associated with the development of carcinoma.

Part of a cæcum with the last few inches of the ileum.

The specimen has been divided so as to show, in section, a lipoma arising from the submucosa. The tumour had caused a thickening of the margin of the ileocæcal opening.

Hunterian Museum, R.C.S., 1273.2

CLINICAL HISTORY.—The patient was a woman, aged 50, who had suffered from constipation for many years. For four months before admission to hospital she had had intermittent burning pain in the right iliac fossa.

On examination the colon was loaded with scybala throughout its length, but after the removal of these the tumour could be felt in the position of the cæcum. The terminal ileum, cæcum, and ascending colon were resected and continuity was re-established by an ileo-transverse anastomosis. There was a gradual improvement in the functions of the bowel.

MULTIPLE ADENOMATA OF INTESTINE.

The upper part of a jejunum opened longitudinally.

Numerous spheroidal tumours hang from the valvulæ conniventes by narrow pedicles.
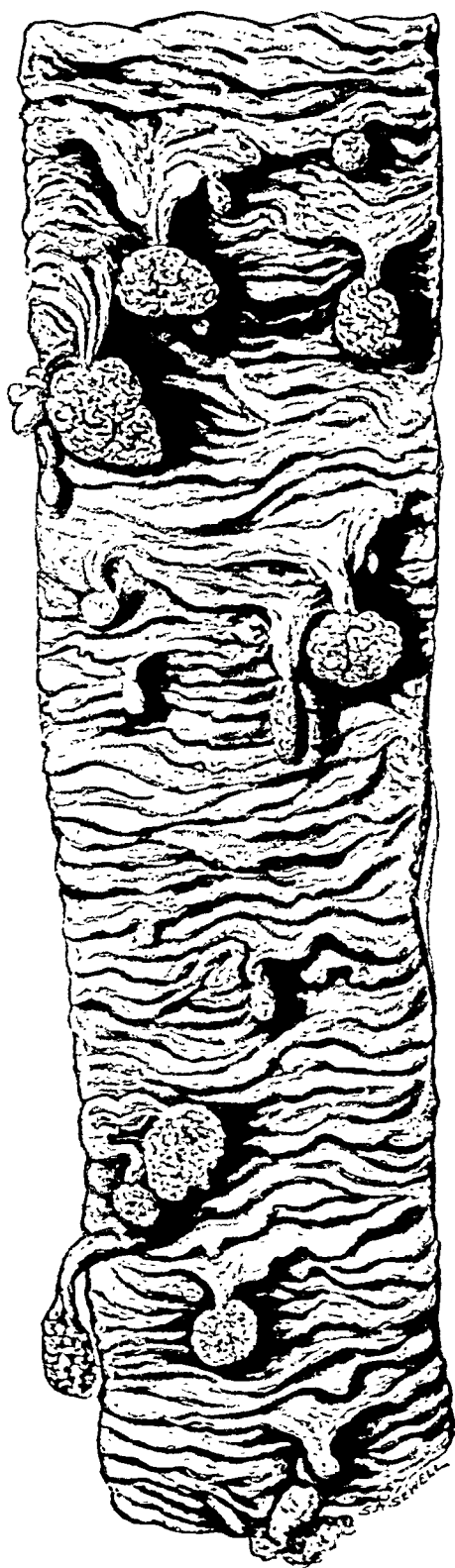
Hunterian Museum, R.C.S., 1512.3

MICROSCOPIC STRUCTURE.—Adenoma.

CLINICAL HISTORY.—The patient was a man, aged 21. who had suffered for twelve months from attacks of severe abdominal pain and vomiting. An intussusception of the duodenum ultimately occurred, and the patient died a few hours after an operation for its relief.

AUTOPSY.—Similar pedunculated growths were found scattered through the stomach, duodenum, and small intestine.

MULTIPLE ADENOMATA OF INTESTINE.

ADENOMA OF SMALL INTESTINE.

Ten inches of small intestine opened along its anti-mesenteric border.

Three lobulated tumours spring from the mucous membrane. the largest being 2 in. wide. Each is attached by a broad base and is smooth on the surface. There is no ulceration.

Hunterian Museum, R.C.S., 6523.1

MICROSCOPIC STRUCTURE.—The tumour is composed of tubular spaces lined by columnar epithelium, with a scanty stroma consisting of small closely-packed oval cells. There is an abundant secretion of mucus from the epithelium.

CLINICAL HISTORY.—The patient was a girl, aged 6 years, who had had occasional attacks of sudden abdominal pain with vomiting for two years. At the end of this time she was admitted to hospital during an attack, and an intussusception was reduced by operation without difficulty. Two weeks later there was a recurrence of the pain and vomiting, and another intussusception was reduced. Five months later she was re-admitted in a similar attack, and the intussusception was reduced by manipulation through the abdominal wall. During the next few months she had several mild attacks which passed off spontaneously. About ten months after the first operation she was again admitted for acute intestinal obstruction associated with a large central abdominal tumour. This time reduction of the intussusception was accomplished with difficulty. During the next six weeks an intussusception was twice reduced by manipulation through the abdominal wall, and at the end of that time a further operation was done at which the tumours were found. The portion of intestine shown was removed and the patient made a good recovery. She was watched for several months and remained free from symptoms.

ADENOMA OF SMALL INTESTINE.

XLVIII.—MULTIPLE POLYPI OF THE INTESTINE.

POLYPI may develop in great numbers in both the small and the large intestines, though they are more often found in the latter. where the condition has received various names—polyposis of the colon, polypoid colon, fibromatosis of the colon.

The disease is originally of a chronic inflammatory nature, but in the large bowel it commonly results in the growth of a carcinoma. The affected portion of colon becomes thickened by the development of fibrous tissue in the submucosa and by the consequent hypertrophy of the muscular coat. Contraction of the fibrosed submucosa produces folds in the mucosa arranged transversely to the long axis of the bowel. These folds, and to a lesser extent the depressions between them, are studded with polypi $\frac{1}{8}$ in. to $\frac{1}{4}$ in. long. Each polypus consists of a core of submucosa covered with mucous membrane. The core is often infiltrated with round cells. Occasionally longer, branched polypi occur. As a rule the mucous membrane is not ulcerated, either over or between the polypi, but it is prone to exude both blood and mucus.

Polypoid colon is a disease which affects both sexes. and develops between the ages of 40 and 50. It is seldom seen in persons under 30.

Multiple polypi of the small intestine cause symptoms, usually by producing an intussusception, at a much earlier age, often between 15 and 20. The polypi arise by short, thick stalks from the ridges of the valvulæ conniventes. Their surface is velvety from the presence of villi. The central core of connective tissue derived from the submucosa is often infiltrated by an overgrowth of lymphoid tissue.

POLYPOID COLON WITH CARCINOMA.

Part of a colon divided longitudinally.

The wall is thickened by overgrowth of fibrous tissue in the submucosa. by thickening of the mucous membrane, and by hypertrophy of the muscular coat. The inner surface is studded with smooth, rounded projections, the smaller ones sessile, the larger ones pedunculated. A few of them are branched. The mucous membrane between and over these polypi is not ulcerated.

An irregular nodular growth projects into the lumen near the centre of the specimen. It is superficially ulcerated and has produced a deep transverse constriction on the outside of the gut. The cut surface shows infiltration of all coats of the bowel, with secondary deposits of growth in the lymph-glands.

Museum of University College Hospital. Alim. G. 19

MICROSCOPIC STRUCTURE.—The polypi consist of a core of connective tissue covered by mucous membrane. Columnar-celled adeno-carcinoma.

CLINICAL HISTORY.—The patient was a man. aged 31. who thought he had had bacillary dysentery in Egypt six years before admission to hospital. Since then his stools had always been loose and he had had much abdominal discomfort. For the last four months the discomfort had increased, and during the last six weeks he had had two attacks of acute abdominal pain. In the first of these he passed blood and mucus, and with the second he had diarrhœa and vomiting and became collapsed.

On examination a tumour was felt above the umbilicus.

At operation there was a carcinoma in the transverse colon to the right of the middle line. The right half of the colon was excised. The patient died five days afterwards.

MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, ALIM. G. 19

XLIX.—TUBERCULOSIS OF THE INTESTINE.

TUBERCULOSIS affects the intestine in two distinct ways: either by the formation of multiple ulcers in the small intestine, or by the production of a single hyperplastic mass in the ileocæcal region.

Multiple tuberculous ulcers are associated with the swallowing of tubercle bacilli, either in milk, or, more commonly, tuberculous sputum in cases of pulmonary tuberculosis. The ulcers are situated where the lymphoid tissue is most abundant at the lower part of the small intestine. Each ulcer is more or less oval in shape with its long axis transverse to that of the gut, and tends to spread round the circumference along the lymphatics. The edge is slightly raised and undermined and the floor is often irregularly pitted. On the peritoneal surface the site of the ulcer is marked by a ring of tubercles. The associated lymph-glands in the mesentery become enlarged. In the process of actual or attempted healing of the ulcers strictures may develop. Extension of the tuberculous process beyond the wall of the intestine leads to the development of tuberculous abscesses and fistulæ and adhesion of neighbouring coils to one another.

Ileocæcal tuberculosis is a primary form of intestinal infection and is not usually associated with pulmonary tuberculosis. The disease commences either in the cæcum or in the terminal part of the ileum immediately proximal to the ileocæcal junction, but in either case tends to spread so as to involve the whole ileocæcal region with its associated lymph-glands.

The tubercles are first deposited in the wall of the bowel and at an early stage can be seen on the peritoneal surface. The wall of the intestine becomes thickened, at first by the tuberculous deposit and later by fibrous tissue as a result of the inflammatory reaction. The thickening of the wall and the contraction of the fibrous tissue cause a progressive diminution of the lumen of the affected portion of the bowel to the point of producing signs of intestinal obstruction. A definite mass is appreciable on clinical examination.

The mucous membrane around the margins of the ulcerated area often develops numerous polypoid outgrowths. Caseation is followed by the formation of tuberculous abscess cavities around the cæcum, and a sinus may open through the abdominal wall.

The appendix may become involved in the tuberculous process, or the obstruction of its cæcal outlet may cause an acute appendicitis, through the treatment of which the true nature of the underlying disease becomes recognized.

TUBERCULOSIS OF INTESTINE.

Part of a small intestine opened longitudinally.

The mucous membrane is extensively ulcerated. The ulcers are arranged transversely to the long axis of the gut. Their edges are slightly thickened and are undermined. Their floors are irregularly pitted. On the peritoneal aspect of the specimen the site of each ulcer is marked by a ring of tubercles.

Hunterian Museum, R.C.S., 857.1

CLINICAL HISTORY.—The patient was a man, aged 32, who died from weakness and diarrhœa after an illness lasting two years.

AUTOPSY.—Tuberculous cavities at the apices of both lungs. Generalized tuberculosis of lymph-glands. Tubercles in liver, spleen, and kidneys. Tuberculous ulceration of duodenum and of small and large intestine. Chronic peritonitis.

TUBERCULOSIS OF INTESTINE
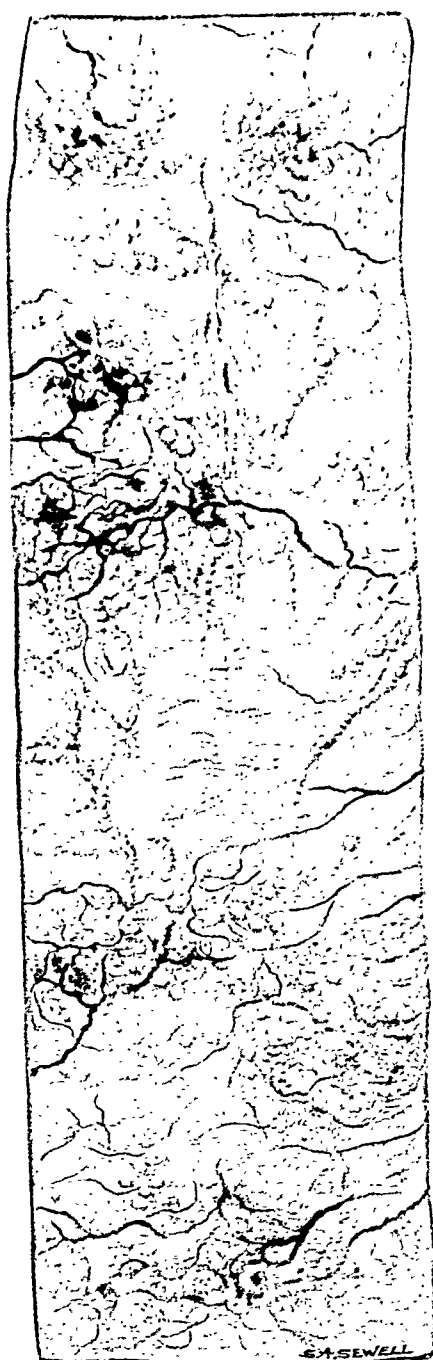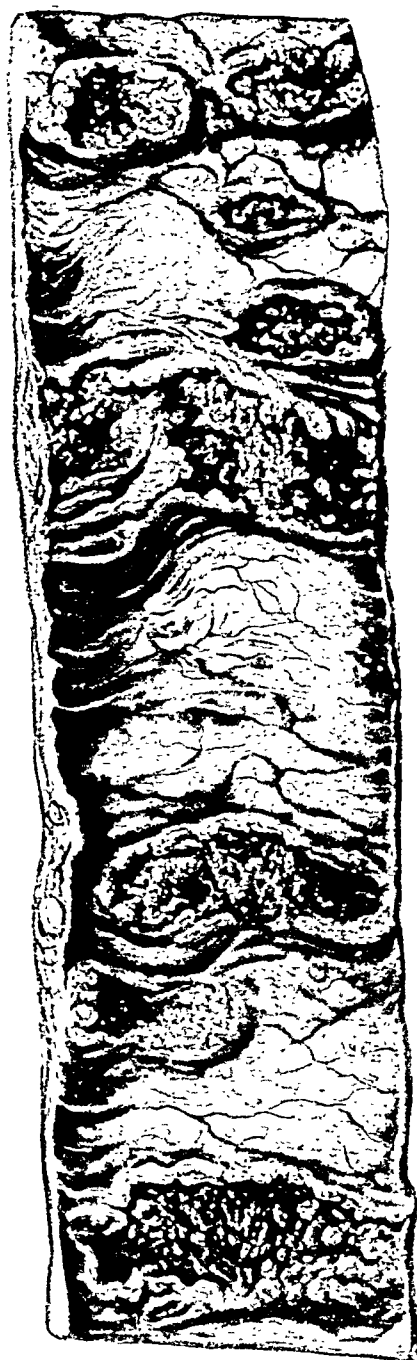
TUBERCULOUS STRICTURE OF INTESTINE.

The last part of an ileum with the cæcum divided by longitudinal section.

The ileum is greatly dilated. In the position of the ileocæcal valve is a dense white mass infiltrating the submucous and subperitoneal coats and causing almost complete obstruction. The mucous membrane is raised into nodules. There is no ulceration. The stone illustrated is one of the many which were present above the obstruction. It consists mainly of calcium phosphate.

Museum of King's College Hospital, E. 217

MICROSCOPIC STRICTURE.—The white mass consists of tuberculous granulation tissue in which are numerous giant cells. There are no miliary tubercles and the degree of caseation is slight.

CLINICAL HISTORY.—The patient was a single woman, aged 20, in whom a cold abscess was opened over the position of the cæcum. The abscess resembled a psoas abscess clinically, though there was no clinical or X-ray evidence of spinal disease. An opaque meal suggested the presence of a fistula between the small and large intestine. After some time a fæcal fistula developed and discharged for ten months. A second X-ray showed great dilatation of the terminal ileum.

The specimen illustrated was excised by operation and the wound healed without delay.

ILEOCÆCAL TUBERCULOSIS.

A cæcum and appendix with the terminal coil of the ileum.

The wall of the bowel is thickened by a deposit of tubercles which are strewn thickly over the peritoneal surface. The mesenteric lymph-glands are enlarged.

Hunterian Museum, R.C.S., 839.2

CLINICAL HISTORY.—The patient was a woman who had suffered from vague abdominal pain and flatulence for five months. During this time there was no diarrhœa or vomiting, but the illness culminated in an attack of subacute intestinal obstruction, after which the temperature was raised to 100°–102° and there was profuse sweating at night. There were no signs of pulmonary tuberculosis.

On examination the abdomen was slightly distended and peristalsis was visible. A tender, nodular, movable mass was felt in the right iliac fossa.

At operation the specimen shown was removed, together with a number of enlarged glands. Ten days later a tuberculous abscess formed beneath the scar and the patient gradually wasted. She died seven weeks after the operation.

ILEOCÆCAL TUBERCULOSIS.
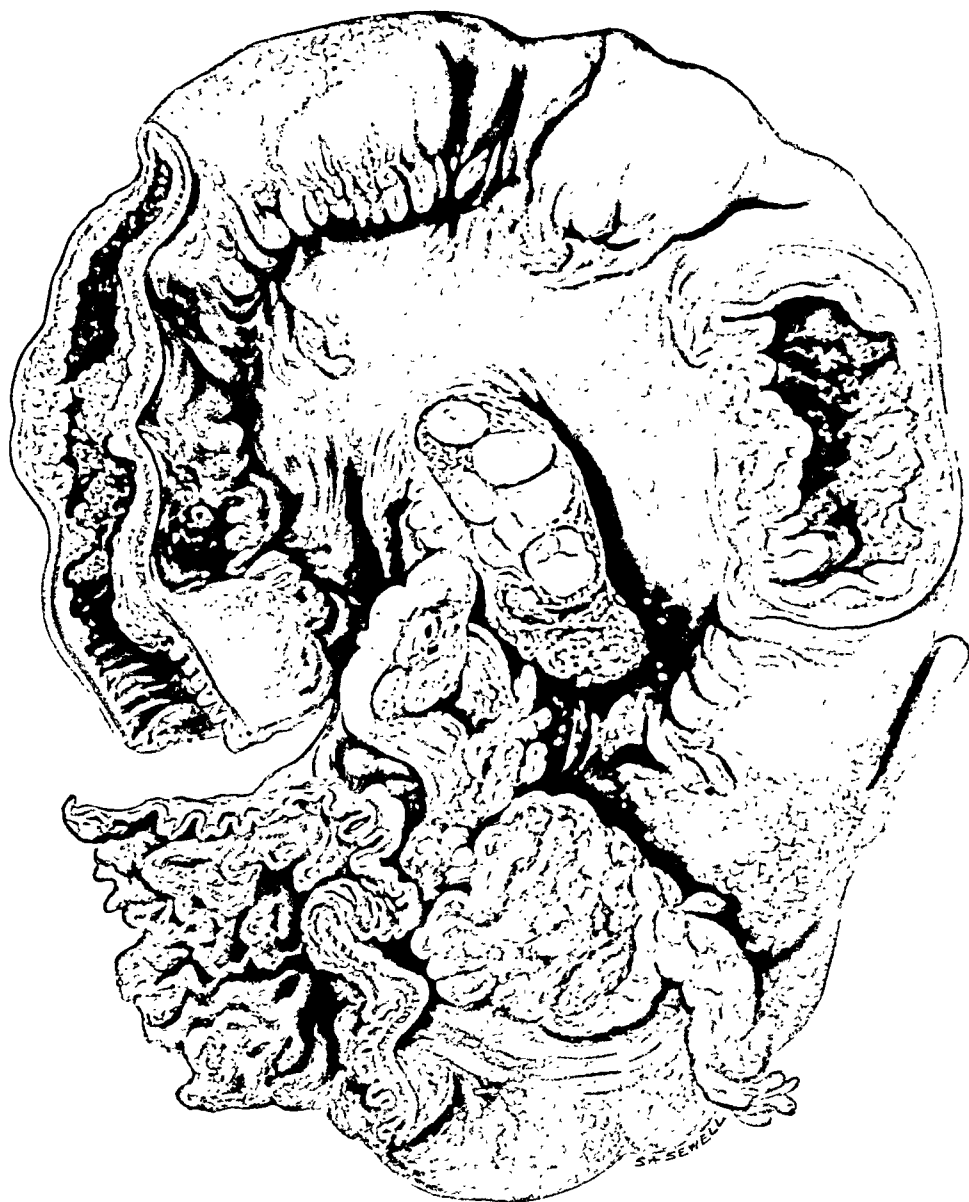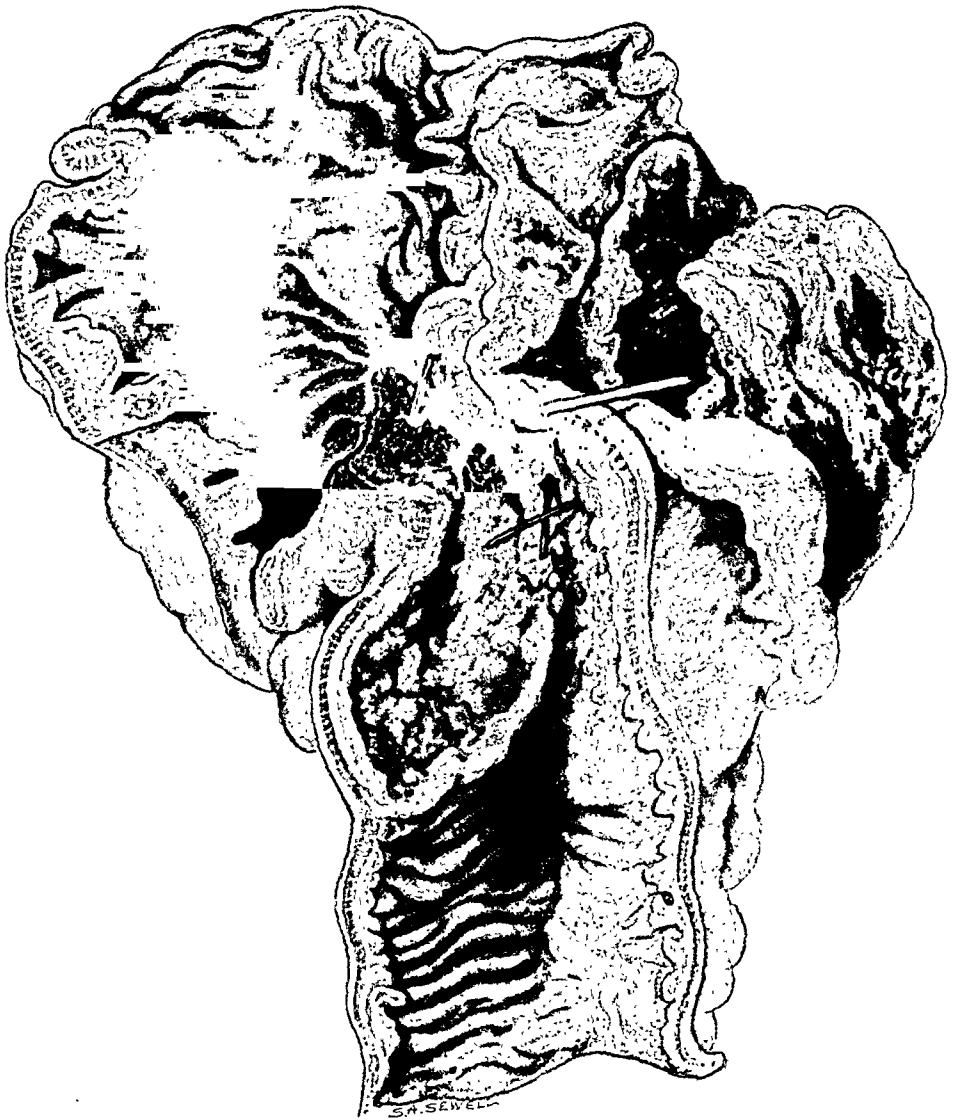
Part of a cæcum with the last few inches of the ileum opened longitudinally.

The small intestine in the neighbourhood of the ileocæcal junction is extensively ulcerated, and from the ulcer two sinuses lead to an abscess cavity on the posterior surface of the cæcum. The lumen of the ileocæcal valve just admitted a probe. The white rod indicates a sinus leading through the abdominal wall to the surface. The lymph-glands behind the cæcum are enlarged.

Hunterian Museum, R.C.S., 858.1

MICROSCOPIC STRUCTURE.—Giant-cell systems in the floor of the ulcer and in the lymph-glands. No caseation.

CLINICAL HISTORY.—The patient was a girl, aged 23, who was admitted to hospital with an abscess in the right iliac fossa of supposed appendicular origin. She had been ill for eleven days. The abscess was opened and a diagnosis of tuberculosis made on account of the glandular enlargement. A sinus remained. The parts illustrated were successfully removed five months later. No after-history.